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CONTENTS

Anterior Poliomyelitis in Massachusetts 1907-1929. *Filip C. Forsbeck* and *Eliot H. Luther.* 1115
Early Diagnosis of Pregnancy: Experience with the Zondek-Aschheim Test. *Benjamin Parvey* 1121
The Patient's Reaction to a Diagnosis of Tuberculosis. *Linsly R. Williams* and *Alice M. Hill* 1129
Two New Clamps for Abdominal Panhysterectomy. *Louis E. Phaneuf* 1131
A Useful Tongue Depressor. *Josiah E. Quincy.* 1132

NEW HAMPSHIRE MEDICAL SOCIETY

Methods and Results of Diabetic Treatment. *Frederick M. Allen* 1133
Pyuria in Children. *Richard W. Robinson* 1139
The Prize Essay. The Prevention of Disease, Some Applications of Preventive Medicine. *Edward O. Otis* 1143
Miscellany 1153

VERMONT STATE MEDICAL SOCIETY

House of Delegates 1154
News Items 1159

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Case 16491—Acute Fever and Precordial Pain. *Richard C. Cabot, George W. Holmes* and *Tracy B. Mallory.* Case 16492—One Year's Intermittent Epigastric Pain. *Richard C. Cabot, George W. Holmes* and *Tracy B. Mallory* 1160

EDITORIALS

Vaccination 1170
A Layman's Interest in Medicine 1170
This Week's Issue 1171

(*Contents continued on next page*)

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HERE is a new text-book that presents histology from its *living, functioning,* and *human aspects.* The student is told not that the kidney elaborates urine, *but which cells of the kidney elaborate which constituents of the urine—and by what mechanism.* At all times Professor Maximow emphasizes the study of *adult tissue,* giving histogenetic data only where they will aid the student's understanding of the mature tissue or organ.

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CONTENTS

(*Continued*)

THE MASSACHUSETTS MEDICAL SOCIETY
Section of Obstetrics and Gynecology 1171
The Committee to Whom Was Referred "The Subject of Preparing Notices of Deceased Fellows" Have Attended to That Duty and Beg Leave to Submit the Following Report 1173

THE BOSTON MEDICAL LIBRARY
Valentine Mott, M.D. 1785-1865 1173

BOOK REVIEWS
The Doctor's Saddle-Bag. Joseph Garland 1180
A Syllabus of Lectures on Obstetrics for Nurses 1180

CORRESPONDENCE
Articles Accepted by the American Medical Association Council on Pharmacy and Chemistry 1176

MISCELLANY
A Criticism of Methods Incident to the Enrollment of Students in Medical Schools 1175
Fund for the Good Samaritan Hospital Reaches Total of $114,099 1175
1930 Seal Sale 1175
The Richness of Milk Is No Guarantee of Its Safety 1175

NOTICES 1177

RECENT DEATH 1176

REPORT AND NOTICES OF MEETINGS 1177

SOCIETY MEETINGS, CONGRESSES, AND CONFERENCES 1179

The Massachusetts Medical Society

The next annual meeting of the Massachusetts Medical Society will be held in Boston, Monday, Tuesday and Wednesday, June 8, 9 and 10, 1931.

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ANTERIOR POLIOMYELITIS IN MASSACHUSETTS, 1907-1929

BY FILIP C. FORSBECK, M.D.,†† AND ELIOT H. LUTHER, M.D.††

ANTERIOR poliomyelitis was made reportable in Massachusetts in November, 1909. Special studies made in 1907[1], 1908[2], and 1909[3] make available fairly reliable figures for those years, so that we are able to base this study on the twenty-three year period, 1907-1929. Cases were reported to the State Department of Public Health; deaths to the State Registrar during the period 1910-1929*. Deaths from poliomyelitis for 1910 and 1911 were determined by special investigation. This study is based on these death and case records, with data relative to age, sex and residence of cases.

TABLE I

ANNUAL INCIDENCE OF CASES AND DEATHS IN MASSACHUSETTS, WITH CORRESPONDING RATES

Year	Cases	Case Rates	Deaths	Mortality Rates	Fatality Rates
1907	234	7.4	*	—	—
1908	136	4.2	*	—	—
1909	923 } †	27.9	*	—	—
1910	845 }	25.0	54	1.6	6.4
1911	260	7.5	36	1.0	13.8
1912	169	4.8	76	2.2	45.0
1913	361†	10.1	69	1.9	19.1
1914	151	4.1	45	1.2	29.8
1915	135	3.6	32	.9	23.7
1916	1927†	51.6	452	12.1	23.5
1917	174	4.6	51	1.4	29.3
1918	99	2.6	37	1.0	37.4
1919	66	1.7	15	.4	22.7
1920	696†	18.0	144	3.7	20.7
1921	233	5.9	48	1.2	20.6
1922	217	5.4	33	.8	15.2
1923	223	5.5	35	.9	15.7
1924	277†	6.8	27	.7	9.7
1925	167	4.0	52	1.3	31.1
1926	245	5.8	44	1.0	18.0
1927	1189†	27.8	169	4.0	14.2
1928	434	10.0	65	1.5	15.0
1929	119	2.7	21	.5	17.6
Totals	9280		1505		
Means	403	10.7	75	2.0	21.5

*Data not available.

GENERAL INCIDENCE

In Massachusetts, poliomyelitis is the most variable of all the common communicable diseases in yearly incidence. The highest incidence recorded is 1,927 cases, the lowest 66. The former is 29.2 times as large as the latter.

Cases, case rates, deaths, mortality rates and fatality rates are listed by years in Table I. During the twenty-three year period under consideration, the mean case rate has been 10.7, the mean mortality rate 2.0, and the mean fatality rate 21.5. The mean number of cases per year has been 403, the median 233. The mean number of deaths per year has been 75, the median 46. There has been an upward trend in the ac-

TABLE II

MASSACHUSETTS INCIDENCE BY YEARS IN ORDER OF MAGNITUDE, WITH SUCCEEDING YEARS

Cases in Order of Magnitude	Succeeding Years 1st	2d	3d	4th
1927	174	99	66	696
1189	434	119	—	—
923	845	260	169	361
845	260	169	361	151
696	233	217	223	277
434	119	—	—	—
361	151	135	1927	174
277	167	245	1189	434
260	169	361	151	135
245	1189	434	119	—
234	136	923	845	260
233	217	223	277	167
223	277	167	245	1189
217	223	277	167	245
174	99	66	696	233
169	361	151	135	1927
167	245	1189	434	119
151	135	1927	174	99
136	923	845	260	169
135	1927	174	99	66
119	—	—	—	—
99	66	696	233	217
66	696	233	217	223

*Previous to 1912 poliomyelitis was not classified as a separate cause of death.

††Forsbeck—Assistant, Rockefeller Institute for Medical Research. Luther—For records and addresses of authors see "This Week's Issue", page 1171.

tual number of cases reported during the period, but there has been little increase in the case rate. Similarly, the number of deaths has been slowly increasing, but the mortality rates have not. The slight difference in the trend of case rate and mortality rate is reflected in a fatality rate trend. Since 1911 the reported fatality rate has been declining at the rate of about 3.4 per cent. per year. This may be due to better diagnosis, better reporting, better treatment, milder disease, or any combination of these. If better reporting is a factor, as it almost certainly is, then the real incidence of poliomyelitis is probably declining, since this better reporting has not been reflected to any appreciable extent in an increased case rate.

There is, of course, a relationship between mortality rate and case rate from year to year, but that this relationship is not very close is shown by the fact that the fatality rate has varied from 6.4 to 45.0. However, this is in great part due to small number errors. It is generally in the low years that the fatality rate deviates markedly from the trend line.

The periodicity of poliomyelitis incidence has frequently been noted. In Massachusetts this periodicity is quite marked. In Table I the six highest years have been marked with an obelisk. The 1909-1910 cases have been considered as occurring between the two years, although we recognize that perhaps it is not fair to do so. Here then are five complete cycles in which the peak years have never been less than three years nor more than four years apart. This general tendency seems to be holding true for the period since 1927. The mean length of periods has been 3.5 years. As would be expected, there is a tendency for the length of period to be longer after the larger epidemics. Both of the smallest periods (three years), followed the smallest two of the peak years.

In Table II are listed cases by years in order of magnitude. After each year's incidence is listed the incidence of the four succeeding years. The blocked-in figures show the number of consecutive years of decrease of incidence. Naturally, a high year may be expected to be followed by a low year. There is, however, a rather striking thing about these figures. The first seven figures in the first column, with one exception, are followed by decreasing incidence for at least two years and the two largest outbreaks for which this information is available, for three years. Data are not yet available, of course, for the period following 1929. In the light of this experience, it seemed quite logical to look for decreasing incidence in 1928 and 1929, and in view of the magnitude of the 1927 outbreak, it seems quite likely that this decrease in incidence will be extended through 1930; 1931 will very likely be a high poliomyelitis year.

TABLE III

ANTERIOR POLIOMYELITIS CASES BY MONTHS, IN MASSACHUSETTS, 1907-1929

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Unknown	Total
1907	1	5	1	5	2	4	12	33	**57**	48	21	16	29	234
1908	1	1	2	1	2	7	**37**	**37**	19	15	9	0	5	136
1909	3	4	5	5	7	34	148	**187**	121	74	17	11	307	923
1910	10	2	2	4	4	22	**159**	157	129	97	46	22	191	845
1911	11	2	9	8	7	8	17	**61**	50	37	16	6	28	260
1912	7	3	8	5	15	12	**34**	28	25	21	10	1	—	169
1913	4	3	4	2	6	1	10	60	**125**	89	38	19	—	361
1914	14	6	8	8	4	11	10	19	25	**31**	9	6	—	151
1915	5	4	6	5	5	4	9	26	15	**28**	16	12	—	135
1916	6	3	5	2	3	11	106	252	623	**701**	179	36	—	1,927
1917	14	2	8	9	9	15	**38**	**38**	16	11	10	4	—	174
1918	5	3	6	6	6	6	10	**20**	**20**	7	7	3	—	99
1919	4	2	2	4	—	3	5	12	9	**17**	5	3	—	66
1920	2	4	4	1	—	5	16	93	**273**	190	77	31	—	696
1921	10	10	7	3	6	4	26	**61**	54	27	15	10	—	233
1922	8	4	4	3	—	4	23	54	**63**	28	21	5	—	217
1923	10	9	5	6	4	4	8	26	38	**48**	40	25	—	223
1924	14	6	9	7	5	5	12	39	**88**	56	23	13	—	277
1925	9	4	7	2	1	2	11	30	**44**	31	14	12	—	167
1926	6	4	5	5	4	5	21	**75**	59	27	26	8	—	245
1927	6	2	3	4	7	11	22	174	373	**376**	146	65	—	1,189
1928	17	13	5	6	7	7	21	**164**	120	50	14	10	—	434
1929	4	3	2	3	8	4	4	11	21	**31**	19	9	—	119
Totals	171	99	117	104	112	189	759	1657	2367	2040	778	327	560	9280
Means	7.4	4.3	5.1	4.5	4.9	8.2	33.0	72.1	102.8	88.7	33.8	14.2	—	—
Percentage Distribution	1.95	1.13	1.34	1.19	1.30	2.17	8.73	19.03	27.16	23.31	8.93	3.76	—	—

MORTALITY RATES AND FATALITY RATES COMPARED

If the mortality rates and their corresponding fatality rates are plotted in order of decreasing magnitude of the former, it may be seen that the fatality rates have a tendency to be higher in

years of high mortality. The factors which influence the computed fatality rate (not the true fatality rate) have been mentioned. Treatment is probably equally good in high and low years, though it may be improving from year to year. Diagnosis and reporting certainly are at least equally good, and very probably better in high than in low years. Therefore, it is logical to believe that the true fatality rate is higher in years of high incidence than in years of low incidence.

SEASONAL DISTRIBUTION

Table III shows the reported incidence of poliomyelitis by months for the twenty-three year period, 1907-1929, with the percentage distribution and the mean for each month. The general shape of the seasonal distribution curve is similar to that given by Aycock[4] for the U. S. Registration Area. The peak is in September, as for that curve, and there is a slight secondary peak in March. The Massachusetts curve varies from the curve for the U. S. Registration Area in that October is the second highest month, on the average, as compared with August for the U. S. Registration Area.

The peak months have been put in bold type. Giving a tie a value of a half point, the peak has occurred in July three times, August six and one-half times, September seven and one-half times, and October seven times. We could discover no relationship between the time of the peak and the size of the year's incidence. There is, however, a tendency in recent years for the peak to occur later in the year than formerly. For example, in the first eleven years, the peak occurred in July or August six times as compared with five times for September or October. In the last twelve years the peak occurred in July or August three times as compared with nine times for September or October. (Allowance was made for the difference in length of months.) Further lapse of time will be necessary to determine whether this is a significant trend.

SEX DISTRIBUTION

The sex distribution of poliomyelitis based on 4048 cases in nineteen years is tabulated in Table IV. On the average, there have been 76.7 cases among females to every 100 cases in males. The variation from the mean has been very small in epidemic years. Only twice during nineteen years have more cases in females been reported than in males, and in these two years the total cases reported for the year have been so few as to permit wide chance fluctuations. There is an increasing relative incidence of the disease in males. In low years the deviation from the mean is greater than in epidemic years.

TABLE IV

SEX DISTRIBUTION OF POLIOMYELITIS IN MASSACHUSETTS, 1908-1929

Year	Males	Females	Total, Sex Known	Number of Females Per 100 Males
1908	77	48	125	62.3
1909	*	*	*	—
1910	331	270	601	81.6+
1911	*	*	*	—
1912	43	47	90	109.2
1913	162	130	292	80.3
1914	52	44	96	84.6
1915	74	56	130	75.7
1916	1075	832	1907	77.4+
1917	*	*	*	—
1918	52	46	98	88.4
1919	29	35	64	120.5
1920	386	293	679	75.8+
1921	139	89	228	64.0
1922	117	92	209	78.6
1923	119	97	216	81.5
1924	169	105	274	62.2
1925	104	61	165	58.7
1926	148	96	244	64.8
1927	657	527	1184	80.2+
1928	246	188	434	76.4+
1929	68	50	118	73.6
Totals	4048	3106	7154	Mean 76.7

*Data not available.

+Note the slight deviations from the mean in epidemic years.

TABLE V

CASES OF ANTERIOR POLIOMYELITIS IN MASSACHUSETTS, 1918-1929

SEX DISTRIBUTION BY AGE

Age	Total	Male	Female	Number of Females Per 100 Males
Under 1	174	87	87	100.0
1	395	232	163	70.2
2	476	281	195	69.3
3	392	216	176	81.4
4	344	193	151	78.2
5	290	162	128	78.9
6	253	141	112	79.3
7	209	111	98	88.2
8	168	102	66	64.6
9	118	65	53	81.4
10	112	65	47	72.2
11	103	54	49	90.6
12	86	44	42	95.4
13	78	41	37	90.1
14	82	57	25	43.8
15	68	47	21	44.6
16	51	33	18	54.4
17	46	30	16	53.3
18	29	18	11	61.1
19	31	15	16	106.5
20-24	110	76	34	44.7
25-29	62	33	29	87.9
30-34	38	21	17	80.8
35-39	32	15	17	113.1
40-44	13	7	6	116.4
45-49	8	3	5	166.7
50-54	7	3	4	133.3
55-59	4	2	2	100.0
60 and over	2	1	1	100.0
Totals	3781	2155	1626	

SEX BY AGE

Table V shows the number of females to 100 males, by age. Unquestionably, at most ages there are a considerably larger number of males affected than females. In all periods of life, except the first year and after thirty, more males are affected than females. Whether the higher ratio of females to males in Massachusetts in the first year of life and the latter part of life is significant, we are not able to state from our figures. The extremely low ratio of females to males in the period of adolescence seems to us to be significant. It is at least interesting that the relative number of females affected during adolescence is unusually low and that this relative number is unusually high at the time of the menopause.

TABLE VI

CASES OF ANTERIOR POLIOMYELITIS IN MASSACHUSETTS BY AGE GROUPS, 1909-1929

Year	0-4	5	6-10	11-20	21-30	31-72	Unk.	Known Ages	Total
1909	408	32	98	46	21	10	308	615	923
1910	345	51	93	69	28	15	244	601	845
1911	(missing)								

Year	0-4	5-9	10-19	20+	Unk.	Known Ages	Total
1912	60	15	8	7	79	90	169
1913	181	58	44	9	69	292	361
1914	62	15	10	9	55	96	151
1915	76	24	17	5	13	122	135
1916	1289	366	110	85	77	1850	1927
1917	(missing)						

Year	0-4	5-9	10-14	15-19	20+	Unk.	Known Ages	Total
1918	63	13	7	5	7	4	95	99
1919	39	13	6	3	2	3	63	66
1920	347	165	67	34	39	44	652	696
1921	98	53	30	13	31	8	225	233
1922	103	52	27	15	10	10	207	217
1923	126	46	20	8	12	11	212	223
1924	126	65	39	15	23	9	268	277
1925	81	27	28	8	17	6	161	167
1926	107	70	23	15	24	6	239	245
1927	536	361	133	70	67	22	1167	1189
1928	160	137	70	30	37	0	434	434
1929	32	38	19	15	11	4	115	119

TABLE VII

PERCENTAGE DISTRIBUTION OF ANTERIOR POLIOMYELITIS IN MASSACHUSETTS BY AGE GROUPS, 1912-1929

Year	0-4	5-9	10-19	20+
1912	66.7	16.7	8.9	7.7
1913	62.0	19.9	15.1	3.0
1914	64.6	15.6	10.4	9.4
1915	62.3	19.7	13.9	4.1
1916	69.7	19.8	5.9	4.6
1917	(missing)			

Year	0-4	5-9	10-14	15-19	20+
1918	66.3	13.7	7.4	5.2	7.4
1919	61.9	20.6	9.5	4.8	3.2
1920	53.2	25.3	10.3	5.2	6.0
1921	43.6	23.5	13.3	5.8	13.8
1922	49.8	25.1	13.0	7.3	4.8
1923	59.4	21.7	9.4	3.8	5.7
1924	47.0	24.2	14.6	5.6	8.6
1925	50.3	16.8	17.4	5.0	10.5
1926	44.8	29.3	9.6	6.3	10.0
1927	45.9	30.9	11.4	6.0	5.8
1928	36.9	31.6	16.1	6.9	8.5
1929	27.8	33.1	16.5	13.0	9.6

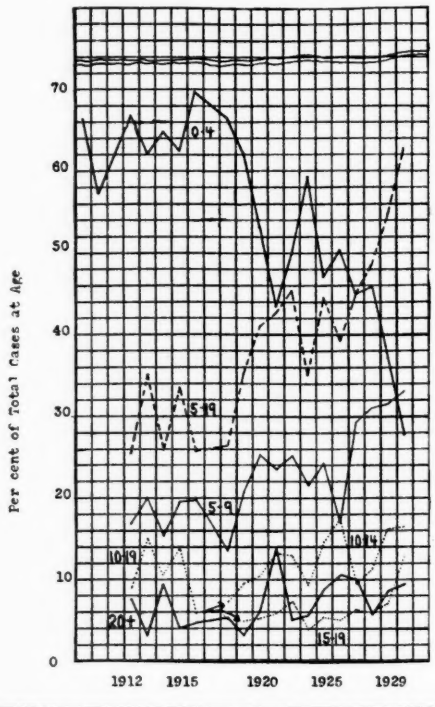
AGE DISTRIBUTION

Table VI shows the age distribution of poliomyelitis in Massachusetts in so far as this information is available. Table VII shows the percentage distribution by age groups for the period 1912-1929 (exclusive of 1917), and for the age group 0-4 for the years 1909 and 1910. The figures of Table VII have been plotted.

Several striking features may be noted in this chart. In the first place, there was apparently little or no change in the percentage age distribution of poliomyelitis up to about 1918. Since then there has been a striking, but unaccounted for, change. The relative number of cases in the 0-4 group has markedly decreased, while there has been a compensatory increase in the relative number of cases in the age group 5-14. The percentage of cases occurring in the age group 15-19 and in adult life has remained constant.

TABLE VII-A

PERCENTAGE DISTRIBUTION OF ANTERIOR POLIOMYELITIS BY AGE GROUPS



Why this change should take place is a mystery. The change in the age distribution of the Massachusetts population is a negligible factor. In the earlier years the number of cases in which the age was not given is large. However, the

trend begins at about 1918, since which time the reporting has been satisfactory as far as age is concerned. Consequently, any systematic error due to this source is negligible in our opinion. Is it within the realm of possibility that persons born during a certain period of years have for some reason a lower resistance?

TABLE VIII

CASES OF ANTERIOR POLIOMYELITIS IN MASSACHUSETTS, MEDIAN AGE IN NINE DENSITY GROUPS

Group	Density of Population*	Median Age
1	0-50	8.1
2	50-100	7.7
3	100-200	6.2
4	200-500	6.2
5	500-1,000	6.0
6	1,000-2,000	4.9
7	2,000-5,000	5.3
8	5,000-10,000	3.7
9	10,000-25,000	4.5

*Per square mile.

Age incidence varies with the density of population. Table VIII shows the median age in nine density groups for a period of fourteen years. It may be seen that in the most sparsely populated group the median age is about eight, whereas in the most densely populated cities the median age is about four. This difference may be accounted for only to a slight extent by the difference in the age distribution of the population.

It may be that because of fewer contacts it takes longer to become infected with poliomyelitis in the country. However, the case rates in the groups studied show no significant difference. From the records it would seem that one is just as apt to get poliomyelitis in the country as in the city, but that it is apt to come later in life.

Tables IX-XIV give populations, cases, case rates, deaths, mortality rates, and fatality rates by density groups for the period 1916-1929.

SUMMARY

1. In Massachusetts there has been little change in the case rate and mortality rate during the past twenty-three years.

2. The relationship between mortality rate and case rate has not been marked.

3. There is apparently a periodicity in the incidence of poliomyelitis and evidently the fatality rate is higher for years of high mortality.

4. The seasonal distribution for poliomyelitis is similar to that for the U. S. Registration Area; the peak occurs in September.

5. There is apparently no relationship between time of the peak and the size of the year's incidence.

6. There is now a tendency for the peak to occur later in the year than formerly.

7. The large number of cases studied shows a ratio of about seventy-seven females to every one hundred males. This discrepancy is even greater during adolescence.

8. There is a significant relationship between age of incidence and density of population, the cases occurring somewhat later in life in rural communities. However, there is apparently no significant difference in the case rate in rural and urban communities.

9. Since 1918 there has been a striking change in the age distribution of poliomyelitis as a whole. The number of cases in the age

TABLE IX

MASSACHUSETTS POPULATION BY CERTAIN GROUPS—1916-1929

	1916	1917	1918	1919	1920	1921	1922
Boston	746,130	746,685	747,240	747,800	751,065	757,080	763,090
100,000-500,000	731,750	743,685	755,630	767,570	777,125	884,600	892,550
50,000-100,000	400,480	404,050	407,630	411,200	413,765	366,440	419,980
25,000- 50,000	578,550	589,085	599,630	610,170	621,202	581,415	540,290
10,000- 25,000	531,110	537,239	553,570	580,505	610,260	633,143	646,312
5,000- 10,000	361,308	359,063	346,950	334,400	324,680	332,462	340,192
2,500- 5,000	178,713	186,144	193,260	190,300	187,640	182,564	189,948
Under 2,500	207,125	202,698	198,222	193,670	194,416	198,039	198,971
State	3,735,166	3,768,649	3,802,132	3,835,615	3,880,153	3,935,743	3,991,333

	1923	1924	1925	1926	1927	1928	1929
Boston	769,100	775,115	781,000	787,000	793,000	799,155	804,840
100,000-500,000	900,500	908,450	916,250	1,024,700	1,033,700	1,042,706	1,051,680
50,000-100,000	474,340	479,340	484,860	389,500	444,750	500,381	557,440
25,000- 50,000	498,340	505,750	564,000	573,400	556,500	514,560	471,157
10,000- 25,000	659,485	692,970	656,243	668,400	675,800	708,955	732,566
5,000- 10,000	353,035	340,850	352,900	380,700	378,500	365,475	372,634
2,500- 5,000	195,003	204,900	207,450	191,840	188,340	197,075	205,753
Under 2,500	197,120	195,138	195,400	198,153	198,693	196,566	184,393
State	4,046,923	4,102,513	4,158,103	4,213,693	4,269,283	4,324,873	4,380,463

Population estimated as of July 1.

TABLE X

REPORTED CASES OF ANTERIOR POLIOMYELITIS BY CERTAIN POPULATION GROUPS
1916-1929

Year	State	(Over 500,000) Boston	100,000 500,000	50,000- 100,000	25,000- 50,000	10,000- 25,000	5,000- 10,000	2,500- 5,000	Under 2,500
1916	1927	491	233	218	369	284	162	75	95
1917	174	13	33	22	53	17	20	8	8
1918	99	14	31	9	13	15	4	7	6
1919	66	13	14	9	5	11	9	2	3
1920	696	191	56	69	161	109	63	18	29
1921	233	47	24	21	52	53	20	8	8
1922	217	47	45	28	18	42	18	7	12
1923	223	40	70	27	23	32	17	3	11
1924	277	56	81	23	32	30	33	15	7
1925	167	39	24	18	27	36	11	6	6
1926	245	13	100	23	30	47	12	9	11
1927	1189	277	215	94	234	168	91	38	72
1928	434	58	64	56	146	42	38	19	11
1929	119	32	24	15	10	18	6	8	6
Total	6066	1331	1014	632	1173	904	504	223	285

TABLE XI

ANTERIOR POLIOMYELITIS IN MASSACHUSETTS—1916-1929
CASE RATES PER 100,000 BY CERTAIN POPULATION GROUPS

Year	State	(Over 500,000) Boston	100,000- 500,000	50,000- 100,000	25,000- 50,000	10,000- 25,000	5,000- 10,000	2,500- 5,000	Under 2,500
1916	51.6	65.8	31.9	54.5	63.8	53.5	44.9	42.0	45.9
1917	4.6	1.7	4.4	5.4	9.0	3.2	5.6	4.3	3.9
1918	2.6	1.9	4.1	2.2	2.2	2.7	1.2	3.6	3.0
1919	1.7	1.7	1.8	2.2	.8	1.9	2.7	1.1	1.5
1920	18.0	25.4	7.2	16.7	25.9	17.9	19.4	9.6	14.9
1921	5.9	6.2	2.7	5.7	8.9	8.4	6.0	4.4	4.0
1922	5.4	6.2	5.0	6.7	3.3	6.5	5.3	3.7	6.0
1923	5.5	5.2	7.8	5.7	4.6	4.9	4.8	1.5	5.6
1924	6.8	7.2	8.9	4.8	6.3	4.3	9.7	7.3	3.6
1925	4.0	5.0	2.6	3.7	4.8	5.5	3.1	2.9	3.1
1926	5.8	1.7	9.8	5.9	5.2	7.0	3.2	4.7	5.6
1927	27.8	34.9	20.8	21.1	42.0	24.9	24.0	20.2	36.3
1928	10.0	7.3	6.1	11.2	28.4	5.9	10.4	9.6	5.6
1929	2.7	4.0	2.3	2.7	2.1	2.5	1.6	3.9	3.3
Mean	10.9	12.4	8.2	10.6	14.8	10.7	10.1	8.5	10.2

TABLE XII

DEATHS FROM ANTERIOR POLIOMYELITIS IN MASSACHUSETTS BY CERTAIN POPULATION GROUPS—1916-1929

Year	State	(Over 500,000) Boston	100,000- 500,000	50,000- 100,000	25,000- 50,000	10,000- 25,000	5,000- 10,000	2,500- 5,000	Under 2,500
1916	452	161	38	61	68	60	30	12	22
1917	51	5	15	3	12	7	5	2	2
1918	37	9	8	3	4	5	1	3	3*
1919	15	3	5	1	0	4	2	0	0
1920	144	47	15	15	23	17	17	4	6
1921	48	13	1	3	8	13	6	2	2
1922	33	11	9	3	5	1	3	0	1
1923	35	9	11	2	6	1	0	3	3
1924	27	6	4	0	6	3	4	1	2*
1925	52	11	5	1	13	8	6	3	5
1926	44	7	16	3	3	12	1	2	0
1927	169	64	36	9	23	13	11	5	8
1928	65	15	10	16	13	3	1	3	4
1929	21	9	3	2	2	2	2	0	1
Total	1193	370	176	122	186	149	89	40	59

*One in Tewksbury State Infirmary not added.

TABLE XIII

ANTERIOR POLIOMYELITIS MORTALITY RATES PER 100,000 BY CERTAIN POPULATION GROUPS—1916-1929

Year	State	(Over 500,000) Boston	100,000-500,000	50,000-100,000	25,000-50,000	10,000-25,000	5,000-10,000	2,500-5,000	Under 2,500
1916	12.1	21.6	5.2	15.2	11.8	11.3	8.3	6.7	10.6
1917	1.4	.7	2.0	.7	2.0	1.3	1.4	1.1	1.0
1918	1.0	1.2	1.1	.7	.7	.9	.3	1.6	1.5
1919	.4	.4	.7	.2	.0	.7	.6	.0	.0
1920	3.7	6.3	1.9	3.6	3.7	2.8	5.2	2.1	3.0
1921	1.2	1.7	.1	.8	1.4	2.1	1.8	1.1	1.0
1922	.8	1.4	1.0	.7	.9	.2	.9	.0	.5
1923	.9	1.2	1.2	.4	1.2	.2	.0	1.5	1.5
1924	.7	.8	.4	.0	1.2	.4	1.2	.5	1.0
1925	1.3	1.4	.5	.2	2.3	1.2	1.7	1.4	2.6
1926	1.0	.9	1.6	.8	.5	1.8	.3	1.0	.0
1927	4.0	8.1	3.5	2.0	4.1	1.9	2.9	2.7	4.0
1928	1.5	1.9	1.0	3.2	2.5	.4	.3	1.5	2.0
1929	.5	1.1	.3	.4	.4	.3	.5	.0	.5
Mean	2.2	3.5	1.5	2.1	2.3	1.8	1.8	1.5	2.1

TABLE XIV

ANTERIOR POLIOMYELITIS

FATALITY RATES IN MASSACHUSETTS—1916-1929—BY CERTAIN POPULATION GROUPS

Year	State	(Over 500,000) Boston	100,000-500,000	50,000-100,000	25,000-50,000	10,000-25,000	5,000-10,000	2,500-5,000	Under 2,500
1916	23.5	32.8	16.3	28.0	18.4	21.1	18.5	16.0	23.1
1917	29.3	38.4	45.4	13.6	22.6	41.1	25.0	25.0	25.0
1918	37.4	64.3	25.8	33.3	30.8	33.3	25.0	42.8	50.0*
1919	22.7	23.0	35.7	11.1	0.0	36.4	22.2	0.0	0.0
1920	20.7	24.6	26.8	21.7	14.3	15.6	27.0	22.2	20.7
1921	20.6	27.6	4.2	14.3	15.4	25.5	30.0	25.0	25.0
1922	15.2	23.4	20.0	10.7	27.8	2.4	16.7	0.0	8.3
1923	15.5	22.5	15.7	7.4	26.1	3.1	0.0	100.0	27.3
1924	9.7	10.7	4.9	0.0	18.7	10.0	12.1	6.7	28.6*
1925	31.1	28.2	20.8	5.6	48.2	22.2	5.4	50.0	83.3
1926	18.0	53.8	16.0	13.0	10.0	25.5	8.3	22.2	0.0
1927	14.2	23.1	16.7	9.6	9.8	7.7	12.1	13.2	11.1
1928	15.0	25.9	15.6	28.6	8.9	7.1	2.6	15.8	36.4
1929	17.6	28.1	12.5	13.3	20.0	11.1	33.3	0.0	16.7
Mean	19.7	27.8	17.3	19.3	15.8	16.4	17.6	17.9	20.6

*One in Tewksbury State Infirmary not added.

group 0-4 has markedly decreased, while there has been a compensatory increase in the relative number of cases in the age group 5-14. The percentage of cases in adult life has remained quite constant.

REFERENCES

1 Lovett, R. W.: The occurrence of infantile paralysis in Massachusetts in 1907. Boston M. & S. J. 159:131-139, July 30, 1908.

2 Lovett, R. W. and Emerson, H. C.: The occurrence of infantile paralysis in Massachusetts in 1908. Month. Bull. Mass. State Bd. of Health, July 1909.

3 Infantile paralysis in Massachusetts in 1909. Month. Bull. Mass. State Bd. of Health, June 1910.

4 Aycock, W. L. and Eaton, P.: The seasonal prevalence of infantile paralysis. Seasonal variation in case fatality. Am. J. Hyg. 4:681-690, November 1924.

We wish to acknowledge the valuable assistance of Miss Helen M. Smith in the preparation of the statistical tables.

EARLY DIAGNOSIS OF PREGNANCY: EXPERIENCE WITH THE ZONDEK-ASCHHEIM TEST*

BY BENJAMIN PARVEY, M.D.†

AT the present time there is no test for the early diagnosis of pregnancy that is universally accepted as satisfactory. Research on this problem, however, has led to a number of contributions and there is promise that a simple and reliable method will be available in the near future. In the experience of the author, the Zondek-Aschheim test, although not entirely simple, has fulfilled the requirements of reliability.

In 1925, Zondek and Aschheim[1, 2, 4, 5, 6] began a series of studies which showed that the hormone of the anterior lobe of the pituitary body was normally excreted in the urine in pregnancy and

*Read before the Harvard Medical Society at the Peter Bent Brigham Hospital, November 25, 1930.

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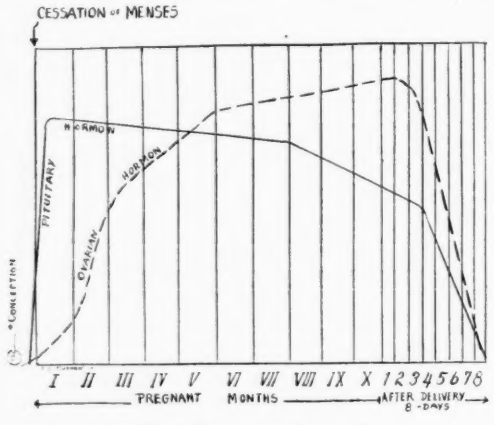
that such urine was capable of producing estrus (bringing the animal to heat). Feeding and implanting[5] infantile mice with anterior lobe of pituitary gland resulted in advanced morphological and functional changes in the ovaries and menstruation occurred approximately 100 hours later. These same changes were regularly produced after the injection into mice of the urine of pregnant women. This finding is the basis of the test which Zondek and Aschheim proposed for the diagnosis of pregnancy. The test, then, is an indirect one and Zondek and Aschheim have shown that other conditions, such as carcinoma, hydatidiform mole and chorionepithelioma increase the formation of anterior pituitary hormone. These conditions, however, are rare.

The action of the pituitary hormone on the ovaries is in contrast to the action of folliculin (ovarian-hormone). Long and Evans[10, 11] and Allen[9] have shown that the latter produces changes confined to the uterine glands and vagina, the structures of the ovaries being unaltered. The mere occurrence of ovarian hormone in the urine is not diagnostic of pregnancy for this is occasionally found in functional disturbances of the ovarian gland, such as may occur at menopause, in certain amenorrheas, hyperthyroidism and myxedema.

The blood of pregnant women shows a marked increase of both pituitary and ovarian hormones (Chart I). With the onset of pregnancy the

CHART I

CURVES ILLUSTRATING CONCENTRATION OF PITUITARY AND OVARIAN HORMONES DURING PREGNANCY



After Zondek and Aschheim. Nr. 6.

The ordinates of this chart are not expressed but the values indicated are based on actual measurements of anterior pituitary and ovarian hormones in urine. The unit for this measurement is the smallest part in cubic centimeters of a liter of urine which is capable of producing estrus in an infantile mouse.

rise of the ovarian hormone is gradual over a period of weeks. In contrast, the pituitary hormone rises rather acutely, a high level being reached a few days after conception. This remains high until the end of the eighth month of pregnancy, after which it gradually drops, reaching normal the eighth day after delivery. The abrupt increase of the pituitary hormone makes possible the recognition of pregnancy as early as five days after an expected period. The value of utilizing pituitary changes other than ovarian as a test for pregnancy is apparent.

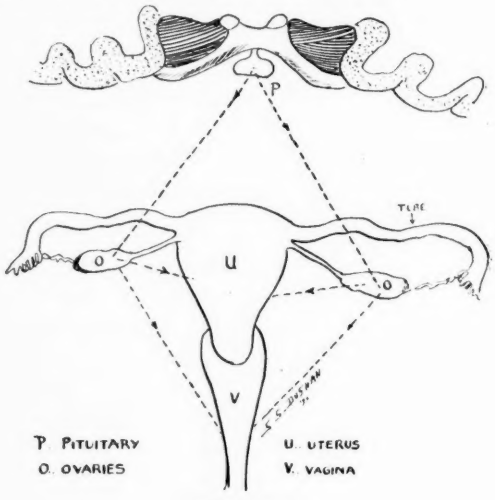


FIGURE I.

Zondek and Aschheim[6, 7] have reported the results of their studies in 511 cases. Of this series 197 were pregnant women, 56 were patients with pelvic disease, 258 were control patients and represented a heterogeneous group of medical conditions in both men and women. There were 12 false reactions. Four of these occurred as positive reactions in control urines—one from a case of cystitis, one from a healthy male and two from patients with carcinoma of the genitalia. Investigation of these showed that the error was due to contamination or faulty technique and a second examination with the same urine specimen gave negative reactions. Four patients with pregnancy gave a negative reaction on the first examination—one at the eighth week, one in the seventh, and two in the ninth months of pregnancy. Second examination of this group was positive in every instance. The error in three of these patients was explained by the fact that in the latter months of pregnancy the urine often contains an insufficient concentration of anterior lobe of pituitary hormone. One case of carcinoma of the cervix of the uterus, one of advanced carcinoma of the ovary, and two cases of hydatidiform mole gave positive tests. The two cases of hydatidiform mole gave negative reactions one week after the uterus was emptied.

Rössler[13] has demonstrated that the anterior pituitary hormone is increased in the urine threefold in hydatidiform mole and in chorionepithelioma. In malignant types of the latter, the increase is from eight to fifteen times the normal amount. He reports results in the study of thirteen cases collected from Stoeckel's clinic. Seven were cases of hydatidiform mole and these gave positive reactions. In six of

these the hydatidiform mole was expelled spontaneously or by curettage and from ten days to six weeks after negative tests were obtained. The remaining patient refused operation and continued to give a positive reaction while being observed for a period of three months. There were three cases of hydatidiform mole and chorionepithelioma combined. These gave negative reactions fifteen to thirty months after extirpation of the uterus. The three remaining cases were cases of malignant chorionepithelioma. One of these gave a negative test two weeks after

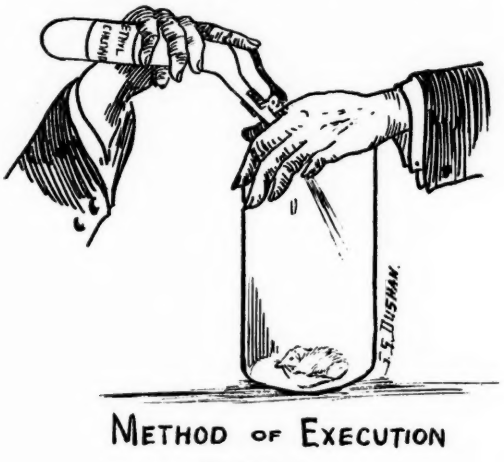


FIGURE II.

hysterectomy; one continued to give a positive test five months after hysterectomy and the third continued to give a positive test a year and a half after hysterectomy. The latter patient died approximately one year and a half after the original hysterectomy and the autopsy showed general metastasis.

On the basis of these three cases, which showed a persistent positive reaction following hysterectomy, Rössler[13], Robert Meyer[14], and Stoeckel[15] recommend the test as a reliable aid in the differential diagnosis of benign and malignant types of chorionepithelioma.

Ehrhardt[16] reported a case of a woman 30 years old, with symptoms of a cerebral tumor, and a history of having expelled spontaneously a hydatidiform mole two years previous. The Zondek-Aschheim test gave a strongly positive reaction even with diluted urine. The high concentration of the anterior pituitary hormone indicated the possibility of a chorionepithelioma. A curettage was performed, but revealed nothing. The patient died, 5 weeks after operation for a brain tumor. Necropsy disclosed chorionepithelioma with generalized metastasis. Fahlbusch[17] and Schultze-Rhonhof[18] applied the test in a group of cases of hydatidiform mole and chorionepithelioma, and found it to be of great value.

Kraul-Rippel[19], Kraus[20], Brühl[21], Ehrhardt[22], Vogt[23], Joffick[24], Kaplan[25], Allan and Dickens[26] and others[27] have carried out the Zondek-Aschheim test in their clinics with 98 to 100% accuracy. In the past twelve months the author has applied this test in the study of 191 patients in private practice*. The results were in full concordance with the claim of Zondek and Aschheim. (Tables I, II and III indicate the author's experience.)

TABLE I

Final Diagnoses	Number of Cases	Reactions II and III Negative	Reactions II and III Positive
1. Pregnancy	79	0	79
2. Metrorrhagia	27	27	0
3. Functional Amenorrhea	38	38	0
4. Menopause	22	22	0
5. Normal	10	10	0
6. Fibromata Uteri	7	7	0
7. Salpingitis	4	4	0
8. Miscarriage	3	2	1
9. Ovarian Cyst	1	1	0
Total	191	111	80

TABLE II

Provisional Diagnoses	Number of Cases	Reactions Negative	Reactions Positive
1. Pregnancy	72	21	51
2. Metrorrhagia	39	27	12
3. Amenorrhea	30	23	7
4. Menopause	21	16	5
5. Normal for Control Test	11	10	1
6. Fibromata Uteri	9	7	2
7. Salpingitis	5	4	1
8. Miscarriage	3	2	1
9. Ovarian Cyst	1	1	0
Total	191	111	80

TABLE III

Period of Pregnancy	Number of Cases	Reactions Negative	Reactions Positive
4-10 days after expected period	35	2	33
2-4 weeks after expected period	29	1	28
3-8 weeks after expected period	14	0	14
3 months after expected period	1	0	1
7½ months after expected period for control	1	1	0
Total	80	4	76

It will be noted that in every case in this series the test checked with the clinical findings. In a few instances false reactions were obtained at first, but these were regularly proven, on repeated examinations, to be the results of error in technique. For example: in three cases the hormone was destroyed by a disinfectant contained in the specimen bottles and in one patient seven and a half months pregnant the afternoon urine was used. It has been shown that in the latter months of preg-

*65 additional cases were examined since this paper was submitted for publication with the same results as tabulated in the charts.

nancy morning urine must be used because of the diminishing concentration of hormone at this period.

Table II gives some idea of the interval elapsing from the time of conception to the time of a positive reaction. These data may be compared with Chart I, which shows the approximate period in which the pituitary hormone begins to rise in the blood. It is reasonable to conjecture that a positive test would be obtained with the first appreciable increase in pituitary hormone and as this occurs very early after conception, the early detection of pregnancy is explained.

It has been shown that the test becomes negative in abortion and ectopic gestation after separation of the foetus from the maternal circulation.

TECHNIQUE

The Zondek-Aschheim test is a biological test and both mice and rats are animals of choice. The former, less expensive, are preferable. The average test requires the use of five infantile mice, three to four weeks old, weighing approximately six to eight grams. Mice weighing less than six grams frequently die before the completion of the test. Mice start the estrus cycle normally about the sixth week.

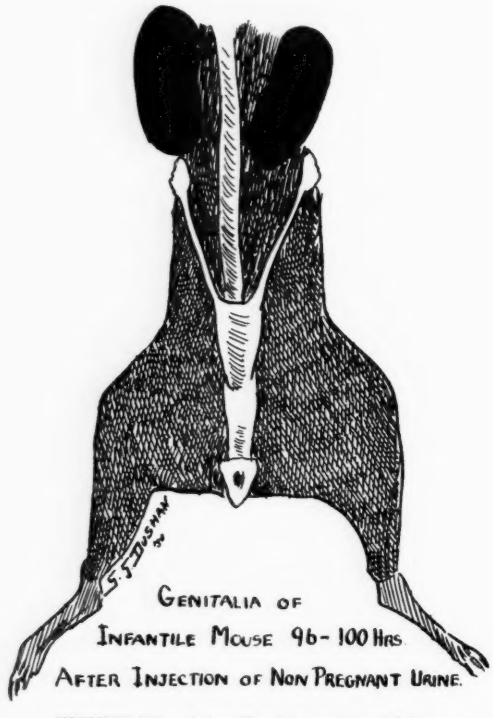


FIGURE III. After Zondek and Aschheim.

The patient's morning urine is chosen because of its greater concentration. It is important that it should be collected in a perfectly clean utensil, for antiseptic drugs, especially perfume, destroy the hormone. When examination is to be delayed, however, one drop of cresol may be added to each ounce of urine as a preservative. Alkaline or neutral urine should be acidified, sediment filtered out if present, and the urine kept in a cool place until used.

The dose injected is conveniently recorded on each mouse by marking with India ink. The animal is grasped by the back of the neck at the level of the ears with the thumb and index finger of the left hand, and, resting it on a table, injection is made subcutaneously in the region of the back. If the mouse scratches or

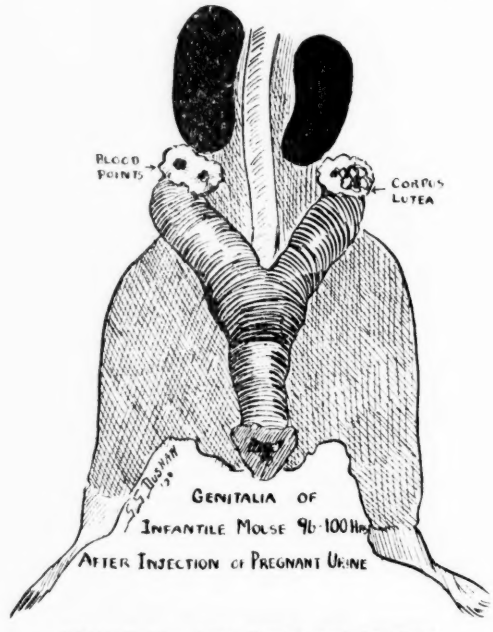


FIGURE IV. After Zondek and Aschheim.

attempts to bite, the tail placed between the middle and ring fingers of the left hand is grasped with the right hand and pulled taut. The urine is injected subcutaneously six times within the first 48 hours: two injections the first day, with five hour interval; three injections the second day, with intervals of three and five hours respectively; one injection the third day on the 48th hour.

Five of the six mice employed to receive injections may be marked respectively on head, back, right leg, left leg and tail for identification. The castrated mouse is left unmarked.

The graded dosages are as follows:

First Mouse	Head	6 injections	0.2 cc.
Second Mouse	Back	6 injections	0.25 cc.
Third Mouse	Right leg	6 injections	0.3 cc.
Fourth Mouse	Left leg	6 injections	0.3 cc.
Fifth Mouse	Tail	6 injections	0.4 cc.
Castrated Mouse		6 injections	0.4 cc.

As already noted, the Zondek-Aschheim test is concerned chiefly with the ovarian changes produced by the pituitary hormone. It is of some value, however, to learn what changes the ovarian hormone is producing at the same time in the uterus and vagina. This will give some index of what might be expected in the ovaries for both hormones, as noted, increased cor-

respondingly in pregnancy and for this reason vaginal smears are taken for examination before the animal is sacrificed.

VAGINAL SMEARS

A smear is made on the afternoon of the third day, on the morning and in the evening of the fourth day, and on the morning before necropsy. The mouse is grasped as mentioned above and placed on its dorsal surface. The vagina is then swabbed deeply with a sterile platinum loop and the contents spread on a glass side. Either methylene blue or eosin is satisfactory for staining.

The findings, 55 hours after injection, have been described as representatives of four stages ([8, 9]) : diestrum, quiescent stage, characterized by the presence of but a few leucocytes, epithelial and mucous cells; proestrum, the proliferation stage, in which a great number of nucleated squamous cells are present; estrus, sexual excitement stage, in which great numbers of squamous cells, some nucleated and non-nucleated epithelial cells are present. The proestrum stage is considered weakly positive, estrus as positive, and the presence of non-nucleated epithelial cells alone is regarded as strongly positive. Metestrus is the building-up stage, and here leucocytes, epithelial and mucous cells are present. The appearance of estrus in castrated mice is, of course, positive.

The castrated mouse is used because the ovarian hormone contained in the injected urines alone will produce estrus. In other mice estrus is produced, in addition, by the ovarian hormone given off from their own ovaries due to anterior pituitary hormone stimulation contained in the urine.

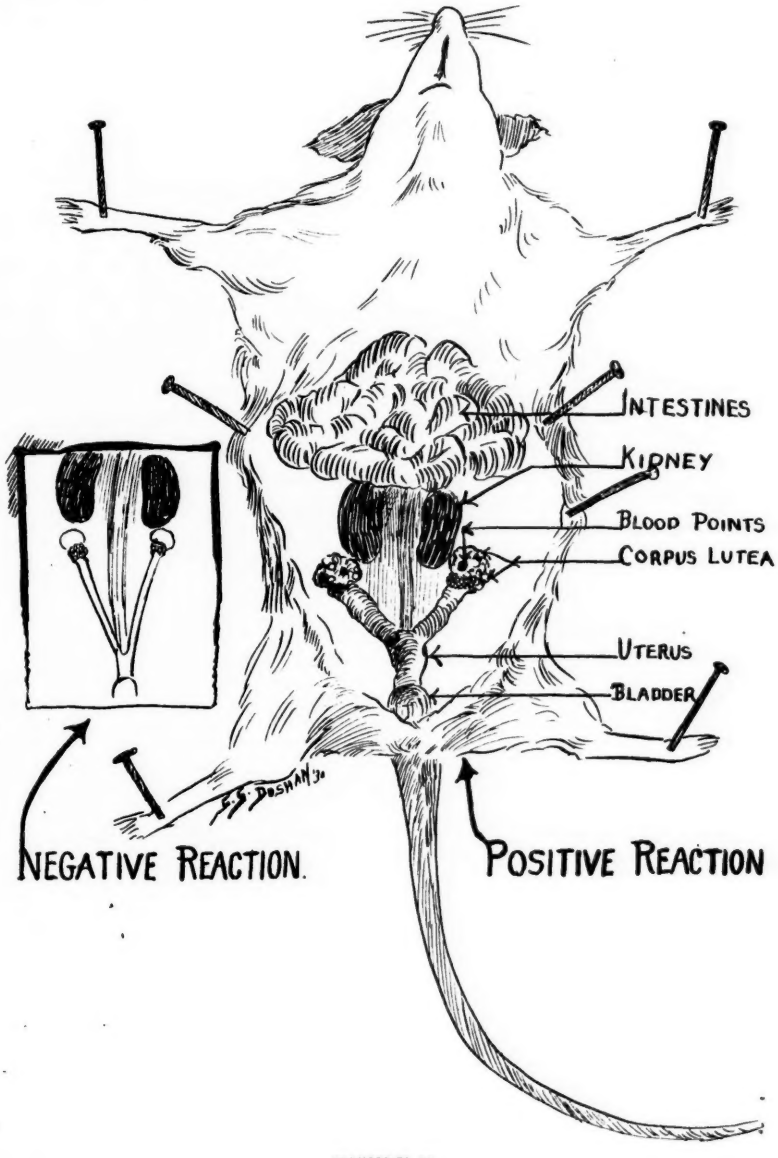


FIGURE V.

Castration: The adult mouse is etherized, placed on its abdomen, and its extremities fixed to the slab. The skin is sterilized, and an incision made over the region of the lower spine, through skin and muscles. The flaps are retracted, and with the peritoneal folds as a guide, the ovaries and periovarian fat are grasped and excised. The peritoneal muscles and skin are sutured with sterile silk. The mouse is kept under observation for 6 weeks before being used as a control test.

Execution: The mice are killed approximately 96-100 hours after the first injections. This may be performed with illuminating gas, ether or chloroform. Ethyl-chloride has been more satisfactory in my experience. Only a small quantity is required and very little odor is produced. The mouse is placed in a small glass jar, the opening covered with the left hand, and ethyl-chloride sprayed between the fingers into the jar. The mouse is usually dead within sixty seconds. The abdomen is opened by a median

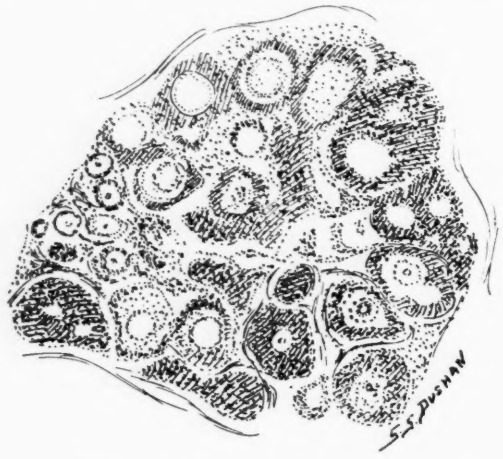


FIGURE VI. After Zondek and Aschheim.

incision from pubis to xiphoid process. The intestines are moved to one side, a horn of the uterus grasped and gently pulled. The peritoneal fat at the lower end of the kidney is removed.

Zondek devised a 48-hour, so called emergency test, which is reliable only when it proves positive. A total of ten injections is given within the first 36 hours.

Criterion of a Positive Test: A positive test is detected by observing gross changes in the ovaries and these have been grouped by Zondek and Aschheim[4, 5, 6] as reactions II and III (figures II and VII). Reaction I in itself is not reliable for the diagnosis of pregnancy as it concerns changes in the uterus and vagina alone. Reaction II concerns the formation of so-called blood-points on the surface of the enlarged ovary. These are discretely distributed, brownish and purplish areas the size of a common pinhead and are the gross representation of hemorrhage into the enlarged graafian follicle. Reaction III concerns the detection of the corpora

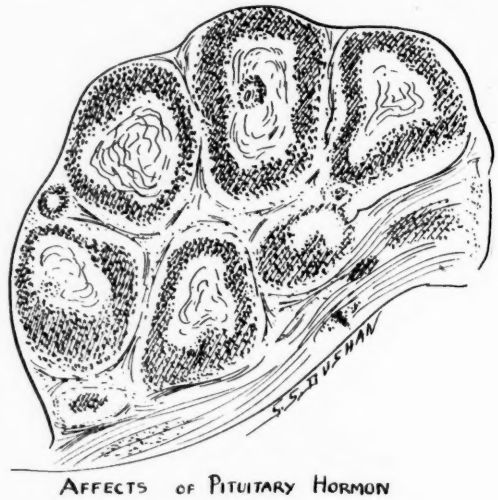


FIGURE VII. After Zondek and Aschheim.

lutea[3, 6, 7, 12], which appear discretely on the surface of the ovary as yellowish pinhead sized areas. The presence of these changes, blood-points and corpora lutea, in an enlarged ovary are diagnostic of an increase in the anterior pituitary hormone and this occurs regularly in

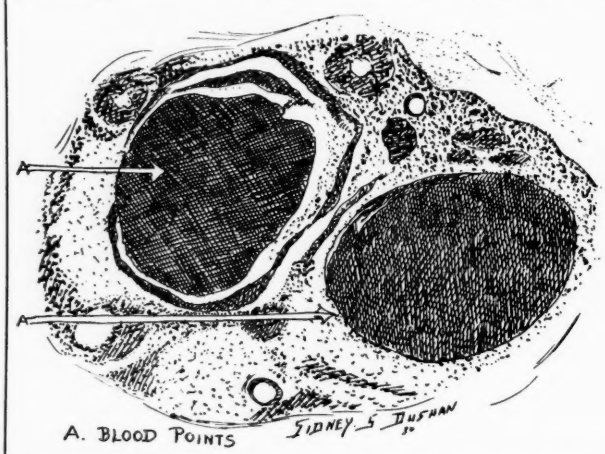


FIGURE VIII. After Zondek and Aschheim—Nr. 6. Blood-points on surface of enlarged ovary.

pregnancy. They may be present in but one ovary.

Sources of Error: There are a few sources of error that can be avoided:

1. It has been shown that the urine at times contains a toxic element which results in premature death of the mice. In the experience of Zondek and Aschheim[6] this occurred in approximately 7.2% of the animals injected. To avoid this difficulty Zondek[7] recommends the following procedure. One part of acidified filtered urine, using a Berkefeld filter, and three parts of ether are mixed and shaken vigorously for 5 minutes. The mixture is placed in a separator.

The urine is drawn off and exposed to the atmosphere near an open window. The ether removes the toxic elements and at the same time the ovarian hormone. The pituitary hormone remains unaltered, in the urine. When the urine is injected, as detailed under *Technique*, characteristic and diagnostic changes in the ovaries are produced. Animals receiving this urine, however, will not develop changes in the uterus and vagina, and vaginal smears will be negative. As emphasized above, this is relatively unimportant, the chief diagnostic characteristic changes being confined to the ovaries.

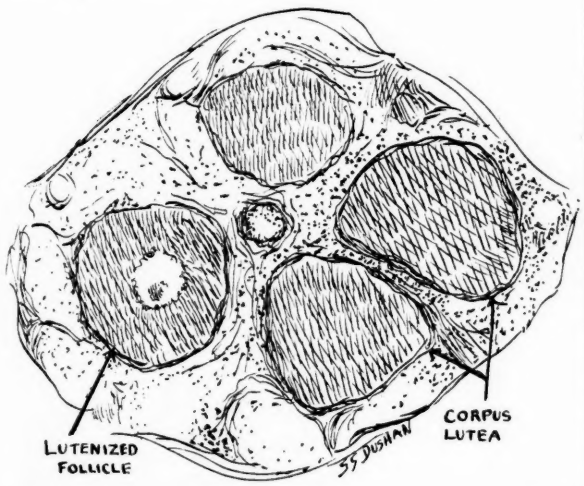


FIGURE IX. After Zondek and Aschheim—Nr. 3. Corpus Lutea on surface of enlarged ovary.

2. The anterior lobe of pituitary hormone is easily destroyed by many household antiseptics and it is of primary importance that utensils used be perfectly clean. The use of perfume bottles has been a source of difficulty in the author's experience.

3. The concentration of the pituitary hormone in the urine is at times so great that an insufficiently cleansed syringe may carry enough hormone with it to produce a falsely positive reaction.

4. To obtain the desired effect of pituitary hormone on the ovaries it is important that the injections be given according to the dose outlined above. Failure to adhere to this routine may, at times, result in a negative reaction, when the same urine properly injected gives a positive result.

5. Urine allowed to remain too long at room temperature may become so overgrown with bacteria as to result in suppuration and premature death of the animal.

6. Accidental injection into the peritoneal cavity may also cause premature death due to rapid peritonitis.

7. Zondek and Aschheim have shown that, in the last few months of pregnancy, the concentration of the anterior pituitary hormone in the urine is often insufficient for the production of ovarian changes. This is particularly true of the afternoon specimen. Zondek and Aschheim have shown that the morning urine is more concentrated and this should be used as a routine.

CONCLUSIONS

1. The technique of the Zondek-Aschheim test for detection of early pregnancy by analysis of urine is presented. Infantile mice are injected subcutaneously with the urine of pregnant women and approximately 100 hours later the mice are examined for characteristic ovarian changes, blood points and corpora lutea. The presence of either of these findings is positive for pregnancy.

2. The accuracy of this test has been investigated by the author in 191 cases. In every instance the findings were in agreement with clinical diagnoses proved by clinical course.

3. Data are presented to show that the test is positive very early in pregnancy. In the series recorded this occurred as early as four days after the expected menstrual period.

4. The Zondek-Aschheim test is recommended as a reliable aid in the diagnosis of pregnancy.

I wish to express my thanks to Professor Zondek and Dr. Aschheim of Berlin, Dr. Harvey Cushing of Boston, Mass., and to the many physicians who so kindly submitted urine specimens for examination.

The following case reports illustrate the value of the test.

CASE REPORTS

CASE I. Intermittent Amenorrhea of Four Years' Duration; Pregnancy Test Positive; Tubal Pregnancy.

A. S., a married woman of 24, was seen April 4, 1930, complaining of excessive menstruation, which had been almost continuous since February 24, 1930. Menstruation started at the age of 12, and was regular with a flow of 6 or 7 days, up to June 1925. From this time to the present date menstruation had occurred but 4 or 5 times, with periods of amenorrhea of 6 to 18 months. Physical examination revealed a uterus somewhat enlarged, the right ovary three times the normal size and tender. On April 5 a D & C was performed and only a few blood clots obtained. Painless bleeding continued. April 14 a second examination was made and a sausage-shaped mass found in the region of the right tube, the right ovary being enlarged, as noted on the previous examination. The history and physical findings suggested a tubal pregnancy. On April 15 a urine examination was instituted. On April 17, 1930 the patient was suddenly stricken with severe lower abdominal pain. She was removed to a hospital and a laparotomy performed. Free blood was found in the abdomen, escaping through the fimbriated end of the right tube. The isthmus of the same tube was greatly swollen but unruptured. Both tube and ovary on this side were removed. The left tube was congested and on its external surface were minute tubercles. The left ovary appeared normal. The pregnancy test proved positive on April 19, 1930.

Pathological report from Doctor Leary's laboratory was as follows: Ectopic pregnancy with hemorrhage and abortion; edema of ovary and chronic and acute periovaritis.

CASE II. Early Pregnancy; Fibroid Uterus; Operation.

D. S., a married woman of 35, multiparous, was seen January 13, 1930, complaining of incontinence of urine. Since adolescence menstruation had occurred at intervals of 6 to 7 weeks. Her last menstrual period occurred November 21, 1929. Physical examination revealed a marked rectocele and cystocele, and a large fibroid uterus. The pregnancy test proved positive. This finding was doubted. A second test was again positive. The patient's request for an operation was refused. She consulted a colleague, who performed a hysterectomy for a large intramural fibroid. Examination of the specimen disclosed a fibroid and in addition a pregnancy of approximately two months' duration.

CASE III. Positive Pregnancy Test 52 Days After Last Period; Negative x-ray Findings 104 Days After Last Period.

E. M. K., a single woman of 37, was first seen March 25, 1930. Menstruation had been regular but scanty since the age of eleven. For the past ten years she had served as a mistress but at no time had taken contraceptive precautions. Her last menstrual period was October 7, 1929. A urine specimen examined on January 10, 1930, was positive for pregnancy. Doubting the laboratory findings, the patient sought information elsewhere and on January 17, 1930, an x-ray was taken and reported negative for pregnancy. Reëxamination by x-ray March 5, 1930, disclosed a skeletal outline. Delivery occurred July 2, 1930.

CASE IV. Pelvic Tumor Suggesting Large Fibroid; Pregnancy Test Positive; Macerated Foetus Delivered Three Weeks Later.

K. S., a married woman of 43, was seen on May 12, 1930, complaining of having failed to menstruate for a period of five months. Menstruation had occurred always at irregular intervals. Physical examination revealed a nodular tumor mass in the region of the uterus. She was examined in two institutions on earlier dates and operation was advised. Urine collected on May 13, 1930, proved positive for pregnancy. On June 6, 1930, the patient went into active labor and was delivered of a macerated foetus approximately five months of age.

CASE V. Metrorrhagia; Dead Foetus; Negative Pregnancy Test.

R. N., a married woman of 40, multiparous, was seen January 12, 1930, complaining of persistent mucous discharge from the vagina of four years' duration. Menstruation was regular up until one year ago, her last menstrual period having been October 5, 1929. Since this time, however, there had been a bloody mucous discharge off and on. She feared cancer. A physical examination revealed a rectocele and cystocele, a lacerated and eroded cervix, a nodular mass 4-6 cm. in diameter at the right side of uterus and a mucoid bloody discharge from the vagina. The uterus was twice the normal size, soft and retroverted. She was ordered to bed for observation. A urine examination for pregnancy proved negative. Bleeding became more and more profuse. On January 19, 1930 a D & C was performed and decayed placental tissue removed.

This case corroborates the experience of Zondek and Aschheim, who have demonstrated that with the death of the foetus the reaction becomes negative.

CASE VI. Pregnancy; First Test Negative; Hormone Destroyed by a sulpho-naphthol Washed Bottle; Second Test Positive.

S. W., a married woman of 31, multiparous, was seen January 22, 1930, complaining of having missed her menstrual period, which was due on December 29, 1929. Urine examined January 9, 1930, for pregnancy was negative. The patient herself, however, doubted the report and on further questioning it was revealed she had washed the specimen bottle with sulpho-naphthol before taking the specimen. A second examination was positive.

CASE VII. Pregnancy Test Positive; Intercourse Denied; Hymen Intact.

R. E., a single girl of 24, on January 25, 1930, offered her urine for a control determination. Menstruation occurred at regular intervals of 5 or 6 weeks and appeared last on December 10, 1929. The test for pregnancy proved positive. Intercourse was denied and examination showed an intact hymen. Reëxamination of the urine was again positive. With the strength of these findings she was induced to admit interfemoral intercourse. Rectal examination showed a uterus one and a half times its normal size. Examination on March 1, 1930, showed a mass corresponding with a pregnancy of 2½ months.

CASE VIII. Amenorrhea; Pregnancy Test Negative.

M. S., a married woman of 27, was first seen February 13, 1930, complaining of having missed her menstrual period, which was due February 5, 1930. She had been nursing a baby nine months of age and during this time her periods had been regular. Urine examinations on February 13 and again on March 8 were both negative for pregnancy. Menstruation occurred normally on March 27, 1930.

CASE IX Amenorrhea; Questionable Increase in Size of Uterus; Pregnancy Test Negative.

B. K., a married woman of 41, multiparous, was seen November 22, 1929. She complained of nervousness and fear of cancer. Her periods had been regular. Her family history revealed that both her father and one brother had died of carcinoma of the stomach, and that a younger sister had been recently operated on for cancer of the uterus. Physical examination showed a marked rectocele and cystocele and a lacerated and eroded cervix. Operation was recommended and when seen three weeks later, she gave the fact that her menstruation was ten days overdue. Local examination showed a uterus somewhat larger than previously noted and suggestive of a six weeks' pregnancy. Urine examination proved to be negative for pregnancy. When a second specimen was requested, she stated that menstruation had occurred three days after the first specimen was submitted.

REFERENCES

1 Zondek, B. and Aschheim, S.: Experimentalle Untersuchungen über die Funktion und das Hormon des Ovariums. Arch. f. Gynäk. 127:250, 1925.

2 Zondek, B. and Aschheim, S.: Der Scheidenzyklus der weissen Maus als Testobjekt zum Nachweis des Ovarialhormons. Technik und Fehlerquellen. Klin. Wchnschr. 5 (pt. 1):979, 1926.

3 Zondek, B. and Aschheim, S.: Das Hormon des Hypophysenvorderlappens; Testobjekt zum Nachweis des Hormons. Klin. Wchnschr. 6:248-252, Feb. 5, 1927; also, *ibid.* Darstellung, chemische Eigenschaften, biologische Wirkungen. Klin. Wchnschr. 7:831-835, April 29, 1928.

4 Zondek, B. and Aschheim, S.: Hypophysenvorderlappenhormon und Ovarialhormon in Harn von Schwangeren. Klin. Wchnschr. 6 (pt. 2):1322, 1927.

5 Zondek, B. and Aschheim, S.: Hypophysenvorderlappen und Ovarium. Arch. f. Gynäk. 130:1, 1927.

6 Zondek, B.: Die Schwangerschaftsdiagnose aus dem Harn durch Nachweis des Hypophysenvorderlappenhormons. Klin. Wchnschr. 7:1404-1411, July 22, 1928; 7:1453, July 29, 1928.

7 Zondek, B.: Schwangerschaftreaktion aus dem Harn durch Nachweis des Hypophysenvorderlappenhormons. Klin. Wchnschr. 9:964, May 24, 1930.

8 Stockard, C. R. and Papanicolaou, G. N.: Existence of a typical oestrous cycle in guinea-pig with a study of its histological and physiological changes. Am. J. Anat. 22:225, 1917.

9 Allen, Edgar: The oestrous cycle in the mouse. Am. J. Anat. 30:297-371, May 1922.

10 Long, J. A. and Evans, H. M.: On the rapid maturation of the ovary by transplants of the youthful gonad to adults. Anat. Rec. 21:60, 1921.

11 Long, J. A. and Evans, H. M.: Oestrus cycle in rats and its associated phenomena. Uni. f. California, 6:1-148, 1922.
12 Sobotta, J.: Über die Bildung des Corpus Luteum bei des Maus. Arch. f. mikr. Anat. 47:261-308, 1896.
13 Rössler, H.: Ueber die Diagnostische Bedeutung des Hypophysenvoderlappenhormons in Urin in Fällen von Blasenmole und Chorionepitheliom. Ztschr. f. Geburtsh. u. Gynäk., Bd. 3.—H—96:516-539, 1929.
14 Meyer, Robert: Beiträge zur Pathologie und Klinik des Chorionepithelioma uteri malignum. Ztschr. f. Geburtsh. u. Gynäk. 92:259-326, 1927.
15 Stoeckel, W.: Verhandlungen der Gesellschaft für Geburtshülelfe und Gynäkologie zu Berlin, Sitzung, Bd. 3-H-96: 640-651, 1929.
16 Ehrhardt, K.: Chorionepithelioma und Schwangerschaftsreaktion. Zentralbl. f. Gynäk. 54:1538-1542, 1930.
17 Fahlbusch: Aschheim-Zondeksche Reaktion und Indikation zur Operation des Chorionepithelioms. Zentralbl. f. Gynäk. 54:1542-1544, 1930.
18 Schultze-Rhonhof: Erfahrungen mit der Aschheim-Zondekschen, Reaktion besonders bei Blasemole und Chorionepitheliom. Zentralbl. f. Gynäk. 54:578-584, 1930.
19 Kraul-Rippel: Experience with Zondek-Aschheim's test of increase of pituitary hormone in urine of early diagnosis. Zentralbl. f. Gynäk. 52:22-26, 1929.
20 Kraus, E. J.: Technik of Aschheim-Zondek reaction for early diagnosis by demonstration of pituitary secretion in urine. Klin. Wchnschr. 8:731-732, April 16, 1929.
21 Brühl, R.: Zondek-Aschheim's test by demonstration of pituitary hormone in urine. Deutsche med. Wchnschr. 55:696-699, April 26, 1929.
22 Ehrhardt, K.: Mechanism of reaction of anterior lobe of pituitary with special consideration of the Aschheim-Zondek pregnancy test by demonstration of pituitary hormone in urine. Klin. Wchnschr. 8:2044-2047, Oct. 1929.
23 Vogt, E.: Diagnosis of pregnancy by demonstration of hormone of anterior lobe of pituitary in the urine. Med. Klin. 25:1725-1726, Nov. 8, 1929.
24 Joffick, S. D.: Female sex hormones and new pregnancy test of Prof. Zondek and Aschheim (demonstration of pituitary hormone in urine). China M. J. 44:24-31, Jan. 1930.
25 Kaplan, H. E.: Aschheim-Zondek hormone test (demonstration of pituitary secretion in urine). California and West. Med. 31:412-413, Dec. 1929.
26 Allan, H. and Dickens, F.: Zondek and Aschheim test (presence of pituitary hormone in urine). Lancet 1:39-41, Jan. 4, 1930.
27 Eino Umfrage von einigen Authoren die Diagnostische Bedeutung der Schwangerschafts Reaktion nach Aschheim und Zondek. Deutsche med. Wchnschr. 56:2125-2130, 1929.

THE PATIENT'S REACTION TO A DIAGNOSIS OF TUBERCULOSIS

BY LINSLY R. WILLIAMS, M.D.,* AND ALICE M. HILL, A.B.*

TWENTY-FIVE years ago Sir William Osler said[1], "There is no greater mistake than to keep from the patient the knowledge that she has tuberculosis in its early stages, as it is only by having that knowledge that she can be expected to recover." At the present day Dr. John Potts writes[2], "There is only one safe rule for both patient and doctor to follow: Diagnose the disease in the early curable stage and then tell the patient that he *is* tuberculous. If this is done the responsibility is definitely placed where it belongs, the patient has a good chance to regain normal or almost normal health and usefulness, *and the doctor has a clear conscience!*" Though both of these statements mention early cases only, indubitably these physicians did not intend to rule out advising patients with moderately advanced and far advanced tuberculosis, as well, of the nature of their illness.

Without definite data available to prove it, the statement is probably quite true that today many more patients are informed promptly that they have tuberculosis than was the case a quarter century ago. Fifty-nine per cent. of the 1,499 patients whose histories were obtained by the National Tuberculosis Association in its study of what happens to tuberculous patients prior to their admission to a sanatorium[3], of which this report is a part, felt quite sure that the physician told them they had pulmonary tuberculosis the very day he made that diagnosis, and an additional 28 per cent., within the first month. On the other hand, 33 patients stated that they were sure a diagnosis of tuberculosis had been made a year or more before they were so informed.

Why should there exist any appreciable amount of hesitancy to tell the patient that he has tuberculosis? How does the patient feel about it? To aid in answering these questions, each patient included in this study was asked his attitude upon being informed that he was tuberculous. Some of the responses were comparatively full, others brief. Not all considered the question from the same aspect, for the question as worded permitted much freedom in the reply. But out of these answers, few of which were couched in the same words, certain definite facts emerge which indicate that from the patient's point of view there is less reason than the physician seems to think for withholding the true diagnosis.

Twelve per cent. of the patients stated that they were not surprised to be told they were tuberculous. Many of these had had tuberculous relatives and were familiar with the symptoms. A number had long suspected the presence of the disease, as was indicated by the following statements:

> "Patient felt she had tuberculosis for nine months before the second physician diagnosed. Called first physician's attention to cough."
>
> "Grateful for the truth and correct diagnosis. Resented the way other doctors passed off her case. Expected the diagnosis of tuberculosis."
>
> "Patient knew she had tuberculosis but could not get her doctor to admit that she had it."
>
> "Patient already felt that he had it. He called the doctor to come through as he felt he was holding out on him. Greatly hurt but wanted to know."
>
> "Had suspected it for many months but could not get first two physicians to tell him so."
>
> "The physician was reluctant to tell the diagnosis, but the patient himself suspected pulmonary tuberculosis and was not alarmed."

Twenty-six per cent. of the patients expressed themselves definitely as appreciative, grateful,

*Williams—President of New York Tuberculosis and Health Association. Hill—Statistical Field Service, National Tuberculosis Association. For records and addresses of authors see "This Week's Issue", page 1171.

relieved to know the truth,—these in addition to 43 patients in the group who had been expecting to be told they had tuberculosis and who placed themselves on record as glad to have the diagnosis settled. Sometimes the patient stated simply that, "she was worried before she knew it; afterwards she picked up"; that "his mind was set at rest"; that "he felt this was the only man to tell him the truth—was really relieved"; that "he figured that the physician did him a favor by telling him he had tuberculosis". Frequently the expression of gratitude was bound up with a feeling of resentment toward the physician who had failed to make the diagnosis or to tell the patient of it. Statements such as these were made: "sore at the first physician because he did not tell her the truth"; "felt badly but felt satisfied to at last find out what was wrong, for all her physicians said nothing was wrong"; "was disgusted that the doctors he had gone to previously had not diagnosed so obvious a condition and he was grateful to the third doctor for his prompt diagnosis"; "did not resent being told but felt resentful at the long time he had to go before diagnosis was made". Many patients also expressed appreciation of the work done by the physicians which led up to the diagnosis. A number of factors contributed to the attitude of another patient whose statement appears worth quoting! "Terribly depressed, but greatly relieved that it wasn't cancer. Satisfied with diagnosis. More hopeful and contented two weeks later because he felt that he could get well. Was 'frantic to know the truth and greatly cheered and relieved to know it was the correct diagnosis'. Was grateful to doctor. Thought first doctor was 'dumb'."

Another 10 per cent. of the patients were quite ready to coöperate with the physician by embarking on such a scheme of treatment as he might prescribe, as probably were for the most part 2 per cent. who stated merely that they accepted the diagnosis.

There appears to have been no reason why the patients in a fourth group should not have been told. These said that the diagnosis of tuberculosis left them more or less unaffected. They comprised 20 per cent. of all those from whom histories were obtained. Some of these patients were so sick that the diagnosis meant little to them. Many were not disturbed because they did not realize the seriousness of the disease. Others were philosophical about it all because the physician explained the diagnosis carefully and told them not to be discouraged.

By 9 per cent. of the patients the diagnosis of tuberculosis was disbelieved, or at least doubted. With this disbelief various other reactions were usually associated. Some of the patients were discouraged or depressed, some were quite willing to take treatment, some were indifferent. In a very few instances the unbelief was in the nature of a defense reaction. But a perusal of the patients' statements leads to the surmise that the truth would have been believed more readily had more thorough examinations preceded the diagnosis and had the nature of tuberculosis been explained more carefully to the patient. In many instances it was reported that the diagnosis was accepted as soon as it had been confirmed by a specialist or by a roentgenological or sputum examination, sometimes the same day. One patient stated that he "didn't believe the first physician and was indifferent. Felt that examination was not thorough enough to justify such an opinion, that the physician simply had that notion. He explained his own symptoms as from bronchitis. Thoroughness of second physician impressed him. When told by this second physician, nearly three years after he saw the first, he was in poor shape and accepted the diagnosis. Did not resent it." In a relatively large number of cases the diagnosis was accepted only after examination in the sanatorium. Of course, a few patients did not think themselves tuberculous because they did not cough or have other symptoms which they recognized as symptoms of this disease; and such patients frequently were not convinced of the truth of the diagnosis until their symptoms had become more pronounced.

The physician is undeniably justified in not telling the patient he is tuberculous when the latter's mental make-up is so abnormal that for him to know the truth would jeopardize his chances of recovery. But the histories of these 1,499 patients indicate that such instances are infrequent. Only five patients stated that when they learned they had tuberculosis they wished they had not been told, but with all five depression in time gave way to resignation, to a more cheerful state, or to a determination to get well. Eighteen per cent. of the patients under consideration were disturbed in varying degree to learn they had tuberculosis. Some said they "felt very badly", were "worried", "discouraged", or "depressed". Others, only about four per cent. of all, reported that they "went to pieces", "gave up every hope", "were greatly frightened", or "collapsed". The eighteen per cent. does not cover all the patients who were more or less upset, but among those it does not include are those who already suspected they were tuberculous or were grateful for the truth even though it was not pleasant. With the great majority any depression sooner or later gave way to optimism or to at least a more philosophical state of mind.

This study indicates that physicians have few grounds upon which to base their reluctance to tell patients they are tuberculous. The argument that to do so will cause the patient to seek another physician is not sufficiently cogent, for, as reported in an earlier article in this series, many more changes were made by these patients

before the diagnosis of tuberculosis was established than afterwards. Doubtless no one is pleased to know that he is tuberculous, but a great many of these patients reported themselves less worried and discouraged when they learned the truth than they were while uncertain what was wrong. Certainly it is the rare patient who when told he is tuberculous will not follow instructions more carefully than the patient who is left in ignorance. Only when advised that his illness is tuberculosis will he be impressed with the need of taking precautions for the protection of others. It is to be expected that the patient will be emotionally disturbed to a certain extent when informed he has tuberculosis, but the histories given by these patients indicate that the degree of depression is frequently a result of the manner in which the character of his illness is explained. When the situation is explained not in blunt but in kindly fashion, not only is the patient more inclined to put his faith in the diagnosis, but the physician is apt to receive the patient's respect, affection, and deep appreciation. This study shows that only in rare instances is it inadvisable to tell the patient that he is tuberculous.

REFERENCES

1 Osler, Sir Wm.: Transactions, National Association for the Study and Prevention of Tuberculosis, 1905, p. 22.
2 Potts, John: Getting Well and Staying Well. C. V. Mosby Co., St. Louis, Mo., 2nd ed., (1929), p. 53.
3 Williams, L. R. and Hill, Alice M.: The Public Health Nurse and Tuberculosis, Public Health Nurse 21: 4-7, (Jan.) 1929; Preventive Medicine as Applied to Tuberculous Patients, J. A. M. A. 92: 774-775, (March 9) 1929; The Utilization of Certain Diagnostic Aids of Special Value in Determining Tuberculosis, ibid. 92: 1989-1992, (June 15) 1929; A Study of What Tuberculosis Patients Experience Prior to Their Admission to a Sanatorium, N. E. J. of Med. 201: 82-91, (July 11) 1929; The Appearance of the Symptoms of Tuberculosis and Their Bearing on the Seeking of Medical Advice, J. A. M. A. 93: 579-582, (Aug. 24) 1929; The Use of "Patent Remedies" by Tuberculous Patients, J. A. M. A. 94: 1292-1294 (April 26) 1930; On Diagnosing Pulmonary Tuberculosis as Another Condition, N. E. J. of Med. 203: 418-421, (Aug. 28) 1930; The Number of Physicians Consulted by Tuberculous Patients and the Reasons for Changing From One to Another, ibid. 203: 782-785, (Oct. 16) 1930; The Loss of Time Before a Tuberculous Patient Is Admitted to a Sanatorium, ibid. 203: 961-969, (Nov. 13) 1930.

TWO NEW CLAMPS FOR ABDOMINAL PANHYSTERECTOMY

BY LOUIS E. PHANEUF, M.D.*

THE question of panhysterectomy versus supravaginal hysterectomy is one about which a great deal of discussion has always centered. There is no gainsaying the fact that the latter is a much simpler operation. On the other hand, it is considered poor surgery, by many operators, to leave behind a badly lacerated and irritated cervix after removing the uterus supracervically. That a certain number of women so treated are exposed to carcinoma of the stump is not to be denied. I have seen five women so afflicted during the course of one year. The removal of the entire uterus is the real prophylactic measure against such occurrence. To be sure, an amputation or a cauterization of the cervix may be performed through the vagina before doing the abdominal subtotal hysterectomy; this overcomes, in a large measure, the irritation of the cervical stump. These procedures, however, prolong the time of operation, and therefore the narcosis, since they necessitate a complete change of drapings, gloves, a second scrub, as well as a change in the position of the patient. These methods do not offer the same protection against cancer that the total hysterectomy does.

The steps which make the total operation a more serious one are, first, the uterine vessels have to be clamped and cut at a lower level, in closer proximity to the ureters and are more difficult to ligate; secondly, the vaginal vessels also have to be ligated; thirdly, the vagina has to be opened below the cervix, which means a lower separation of the urinary bladder and more difficulty in suturing the vaginal cuff than is encountered in closing the stump of the cervix.

In order to overcome these difficulties, I have had two clamps made which have simplified these two important steps.

1. The clamp for the uterine vessels (the lower one in the illustration) is 8½ inches in length, has a short jaw with a toothed point and makes an obtuse angle with the shaft. By the use of this clamp, especially in a deep pelvis, a ligature is placed around the uterine vessels with the greatest ease. In my experience this forceps has never slipped and there has been no retraction

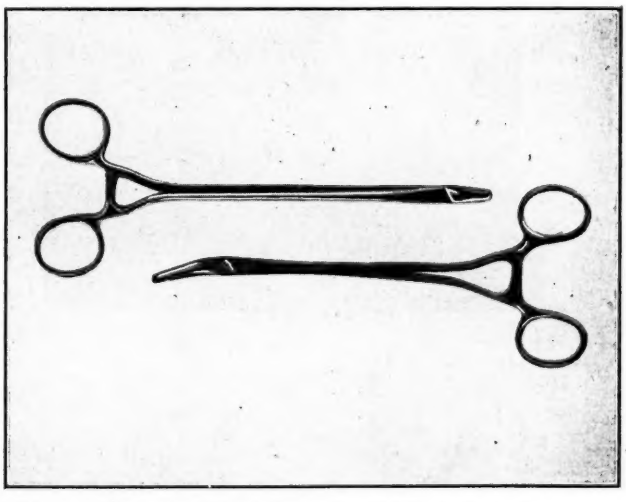


FIG. 1. Clamps for panhysterectomy. Upper clamp in illustration has a short straight jaw and a toothed point. It is used to hold the edges of the incised vaginal cuff.
Lower clamp in illustration has an angulated short jaw and a toothed point, and is used to clamp and ligate the uterine vessels.
Both clamps are 8½ inches long and have box locks.

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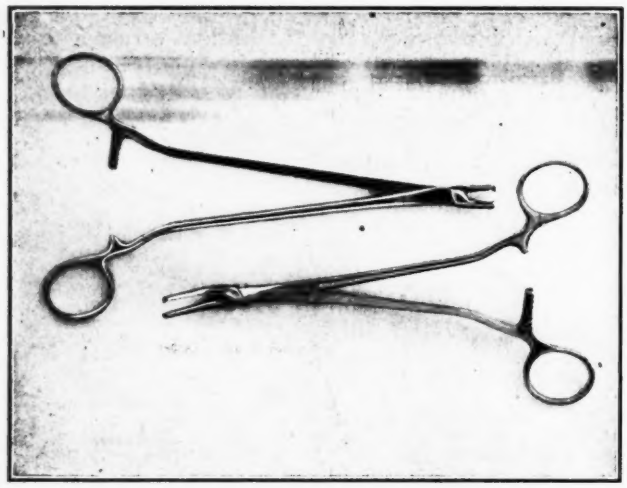

FIG. 2. The same clamps with the jaws opened.

of the vessels. This instrument may also be used to clamp the uterine vessels in doing a supravaginal hysterectomy.

2. The vaginal clamp (the upper one in the illustration), is 8½ inches in length, has a short jaw with a toothed point so that it holds the vaginal cuff without tearing. The handle is long enough to be out of the way during the approximation, by sutures, of the anterior and posterior vaginal walls.

Formerly I used Ochsner clamps, which were too short for a deep pelvis; then Jacobs' volsella, which generally tore the edges of the vagina during the suturing. These complications have been entirely overcome with the use of the forceps described.

Both of these clamps have box locks and since they are used for but one purpose, there is very little chance of their getting out of order. By their use I have found the performance of panhysterectomy very much simplified.

A USEFUL TONGUE DEPRESSOR

BY JOSIAH E. QUINCY, M.D.*

THE illustration (A) shows a child size metal tongue depressor, which has been modified from the adult size, (B). The advantage of this instrument can be appreciated only by using it. Obviously, a tongue depressor which is in proportion to the size of the child's mouth allows more room to operate than does one of adult size. The dimensions are: the handle 4½ inches long, ½ inch wide, the tongue 2½ inches long, widening from ½ inch to ¾ inch at the tip.

A B

A B

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NEW HAMPSHIRE MEDICAL SOCIETY

METHODS AND RESULTS OF DIABETIC TREATMENT*

BY FREDERICK M. ALLEN, M.D.†

THE rational treatment of any disease must be guided by a conception of its cause and nature. Though by no means everything is clarified regarding diabetes, the strongest evidence indicates that it is a disorder of the pancreas and its nature may be defined as a deficiency of insulin. We may assume that the starting point of every typical case is some infection or intoxication, frequently acute in character, the pancreatic damage becoming manifest in the form of diabetes either immediately or months or years later. Two predisposing causes also appear to be influential, namely, heredity and overeating or obesity. By these influences we can explain the high incidence of diabetes in some races, families or classes of population, but it still seems probable that injury to the islands of Langerhans by infection or intoxication must be the direct starting point in practically all cases.

In addition to this primary pathology, there is a secondary pathology, namely, a pancreatic lesion resulting from the diabetes. This is the so-called hydropic degeneration of the island cells, first described by Weichselbaum in 1901. My own researches showed that this is not on a par with fibrous, hyaline, fatty and other primary changes, but, on the contrary, is an exhaustion and degeneration of cells from overstrain of their internal secretory function. In partially depancreatized animals the hydropic change can be halted by checking the diabetes. The loss of cells through hydropic degeneration causes the islands to diminish in size and number, and is largely responsible for the scarcity of islands found at autopsy in many severe diabetic cases. We thus have an anatomical explanation of the tendency of diabetic cases to become worse if glycosuria is allowed to continue, and of the necessity of thorough control of the sugar in order to prevent downward progress. Contrary to the old tradition, diabetes is not an inherently progressive disease; it is progressive only under two harmful influences, namely, infection or inadequate treatment. Hydropic degeneration has become rare nowadays in comparison with former times, because it is prevented by insulin treatment.

If diabetes is a deficiency of insulin, we have logical treatment indicated in two forms: first, to reduce the need for insulin by dietary regulation, and secondly, to give additional insulin if necessary, now that this extract has become available. I shall discuss first and chiefly diet treatment, as it was the earlier one and as it still remains the chief ground of confusion and dispute.

DIET TREATMENT

So many contradictory opinions and fads are promulgated in regard to diet that the average practitioner must be considerably confused by them. From the former advocacy of very high fat rations the pendulum now tends to swing to the opposite extreme of excessively high carbohydrate. In 1914 I introduced a diet treatment which emphasized mainly the restriction of total calories and body weight. This principle was based firmly upon animal experiments, and it furnished the means of clearing up both sugar and acetone in a large class of cases of severe diabetes which had previously been uncontrollable. Numerous and conclusive clinical experiments were published, showing that reduction of the total calories and body weight tends to abolish diabetic symptoms and raise the tolerance, while increase of total calories and body weight tends to reduce the assimilation and bring back glycosuria and other symptoms. This principle was proved to hold good when the increase of calories and weight was produced by means of fat[1, 2, 3], which had previously been regarded as harmless, or by alcohol[4], which was formerly viewed as an ideal food in diabetes because it does not directly give rise to either sugar or acetone. After the discovery of insulin these experiments were repeated, with the modification that the changes in tolerance could now be directly measured in terms of isulin dosage, and the principle of total caloric regulation was strongly and completely confirmed by the demonstrable changes in insulin requirement[5, 6]. These experiments have never been overthrown or even attacked. Some writers have simply ignored them and have undertaken to replace undernutrition with excessive fat diets, thus reverting to the old error of the harmlessness of fat, which had been thoroughly exploded. Other evidence consisted only in the vague observation that a series of patients, mostly with mild forms of diabetes, could live for considerable periods on diets containing excessive proportions of fat. It must also be added that any prolonged control of genuinely severe cases was still accomplished only by the aid of undernutrition, as reference to the publications of the advocates of high fat diets will readily prove. Now that

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insulin has made possible a greater liberality in diabetic diets, there is a strong swing in the direction of high carbohydrate allowances. The proponents of this plan are entirely correct in their observation that liberal carbohydrate sometimes makes patients feel better and, on the whole, requires surprisingly little increase of insulin dosage, provided always that the limitation of total calories and body weight is properly maintained. All the observations with extremes of diet in either direction, so far as they are correct, fall within the scope of the principle which was established by my experiments mentioned above.

To illustrate these statements, I am showing this evening three lantern slides of hydropic degeneration in the pancreas and two dozen slides of tables illustrating the effects of experimental changes in the various components of the diet and in the body weight of diabetic patients. Only one table of this kind will be reproduced in print here. Anyone interested in further details can find a sufficient series of such experiments by referring to the earlier publications cited above. Furthermore, it is not necessary to take anybody's word for these facts, because repetition of the experiments is easy for any physician who keeps patients on accurate treatment. The results are most easily and clearly seen in the severest cases, because these are most readily affected, but with sufficiently long observations the same effects can be demonstrated in cases of any grade. In fact, the improvement of tolerance by reduction of the weight of an obese patient with mild diabetes is one of the very common and important applications of this principle in practical therapy.

The table shown here is part of the record of a severely diabetic Jewish woman, aged 34 years, 5 ft. 5⅜ in. in height. Heavy glycosuria and also heavy acetonuria were present. With a very low diet of 30 gm. protein, 5 gm. carbohy-

Date—1923	Diet P.—Gm.	Diet F.—Gm.	Diet C. H.—Gm.	Diet Cal.	Glycosuria—Gm.	Plasma Sugar—Mg. per 100 cc.	Weight—Lb.	Insulin—Units
Jan.								
22	30	15	5	275	25.20	429	97	0
23	"	"	"	"	24.96	.....	.....	0
24	"	"	"	"	17.54	366	.....	0
25	"	"	"	"	3.50	.....	99	2
26	"	"	"	"	1.80	357	.....	2
27	"	"	"	"	+	.....	98	2
28	"	"	"	"	0	.....	.....	2
29	"	"	"	"	+	.....	97	2
30	"	"	"	"	0	288	.....	2
31	30	18	5	"	0	.....	95	2
Feb.								
1	30	15	75	555	0	268	.....	2
2	"	"	"	"	6.48	.....	.....	2
3	"	"	"	"	3.17	.....	94	2
4	"	"	"	"	6.16	.....	.....	2
5	"	"	"	"	6.72	375	90	2
6	"	"	"	"	18.90	.....	.....	2
7	"	"	"	"	+	366	89	4
8	"	"	"	"	2.10	.....	.....	4
9	"	"	"	"	0	.....	92	6
10	"	"	"	"	0	217	89	6
11	"	"	"	"	0	..... / 254*	.....	6
12	"	"	"	"	0	220 / 230*	89	6
13	"	"	"	"	0	.....	.....	6
14	"	"	"	"	0	200 / 217*	90	6
15	"	"	"	"	0	214 / 312*	.....	6
16	"	"	"	"	0	218 / 300*	89	6
17	"	"	"	"	0	..... / 270*	.....	6
18	"	"	"	"	0	238	87	6
19	"	"	"	"	0	.....	.....	6
20	"	"	"	"	0	.....	86	6
21	"	"	"	"	0	270 / 238*	.....	6
22	"	"	"	"	0	250 / 129*	87	
23	"	"	"	"	0	234 / 211*	.....	6
24	"	"	"	"	0	.....	88	6
25	100	6	25	555	0	.....	.....	6
26	"	"	"	"	0	150	90	6
27-28	"	"	"	"	0	.....	89	6
Mar.								
1	"	"	"	"	0	135	.....	6
2-4	100	6	100	855	0	.....	89	6
5	"	"	"	"	0	211	.....	6
6	"	"	"	"	0	.....	90	6
7	"	"	"	"	0	211	.....	6
8	"	"	"	"	0	203 / 214*	90	6
9	"	"	"	"	0	.....	.....	6
10	"	"	"	"	0	..... / 124*	90	6
11	"	"	"	"	0	214	.....	6
12	"	"	"	"	0	.....	90	6
13	"	"	"	"	0	187	.....	6
14	80	206	80	2500	0	.....	89	6
15	"	"	"	"	0	.....	.....	6
16	"	"	"	"	+	.....	90	6
17	"	"	"	"	+	300	.....	6
18	"	"	"	"	3.64	.....	89	6
19	"	"	"	"	15.72	.....	.....	6
20	"	"	"	"	33.00	.....	91	6
21	"	"	"	"	37.20	375	.....	6
22	"	"	"	"	28.68	.....	93	6
23	"	"	"	"	27.72	375	.....	6
24	"	"	"	"	24.50	.....	92	6
25	"	"	"	"		385	.....	12

Plasma sugars ordinarily taken before breakfast and before insulin in the morning.

*Indicates blood taken at 7 P. M.

drate and 275 calories there was no immediate cessation of glycosuria, but the cessation was obtained with the aid of only two units insulin per day. On February 1, the protein and fat were left unchanged and the carbohydrate was increased by 80 gm., a moderate increase of calories thus resulting. Glycosuria followed, but it was small in proportion to the carbohydrate addition and was checked by a small increase of insulin (up to 6 units daily), because the diet was still one of undernutrition. On February 25, the protein was increased to 100 gm. and the carbohydrate reduced to 25 gm., while the calories were kept unchanged. No harmful or toxic effect of the protein was evident, but on the contrary the blood sugar fell appreciably in consequence of the lower carbohydrate. Beginning March 2, the 100 gm. protein was continued, while the carbohydrate was raised to 100 gm., thus increasing the total calories by 300. The blood sugar rose, but glycosuria remained absent because the great increase of carbohydrates was largely counterbalanced by the prolonged undernutrition. The actual loss of body weight in this process was partly masked by the usual water retention. On March 14, the protein and carbohydrate were each reduced to 80 gm., but the total calories were raised to 2500 by a large addition of fat. Glycosuria resulted and was not stopped by increasing the insulin to 12 units daily. Subsequently this patient, at a weight of 104 pounds, required 20 units of insulin per day to keep her sugar-free.

This single illustration represents relatively small changes in diet, body weight and insulin. Other experiments previously published offer a variety of illustrations, including the effects of extreme dietary changes and more prolonged observations. Carbohydrate is obviously the most powerful in producing glycosuria, but not to the degree which would be expected by those who imagine that carbohydrate is the only offending substance in diabetes. Quantities of protein up to 300 gm. daily have shown no toxic or specific injurious effect upon the tolerance. Protein produces slightly more glycosuria than an equal number of calories in fat, but scarcely as much in general as would correspond to its theoretical carbohydrate equivalent (56% of its weight). Fat has little immediate or direct influence, but when used as a means of building up excessive calories and body weight, it produces effects out of all proportion to any theoretical glucose equivalent, and under these conditions it produces the greatest of all increases of glycosuria and insulin requirement.

Some of our unpublished experiments extend over two years, and in some phases show the following: A typical patient may have an insulin requirement of about 40 to 50 units daily, with any reasonable variation of protein and carbohydrate, while the body weight is kept normal by suitable limitation of the total calories. But when the total calories are greatly increased by addition of fat so as to produce obesity, the insulin requirement rises perhaps to 100 or 150 units daily. A return to the original low dosage is then not obtainable by any reductions of carbohydrate or protein but only by reduction of the total calories and body weight.

One effect of this demonstration is to explode the idea of attempting to reckon diets merely in terms of carbohydrate equivalents. It also exposes the pseudoscientific calculation of ketogenic-antiketogenic ratios. The purpose of this proposed calculation has been to supply as many calories as possible in fat, together with the minimum of carbohydrate and protein that will suffice to prevent acidosis. This goes back to the old fallacy that fat is harmless and only carbohydrate need be considered as taxing the tolerance. In actual fact, fat is not harmless but must be limited with a view to the patient's tolerance. The theoretical glucose content of the diet need not be held at a minimum but may be varied considerably in the form of both carbohydrate and protein without greatly changing the insulin requirement, so long as the total calories are properly regulated. The fallacy of constructing diets on these ketogenic calculations is all the worse because it was originally promulgated in defiance of complete proof which already existed against it.

We still have no real knowledge of the function of insulin in the body. Possibly it is necessary for the normal metabolism of all food substances, or is concerned in some phase of intermediary metabolism in which derivatives from all classes of food participate. For example, it is conceivable that a totally depancreatized dog should be able to nourish himself for a considerable time with fat and the noncarbohydrate moiety of protein. But though he burns these substances, they do not maintain his nutrition or life, even though some insulin is still present in his organs. It is, therefore, a possible and plausible hypothesis that insulin is indispensable for normal metabolism in general. On the other hand, it is still possible that insulin takes a direct part only in carbohydrate metabolism, but that the utilization of carbohydrate is made more difficult by the simultaneous utilization of other food substances. Inasmuch as there is nothing resembling a ratio between the carbohydrate of the diet and the insulin requirement, there has been some tendency to suggest that the insulin need is related to the total metabolism. This supposition apparently holds good for the increased insulin requirement which usually accompanies the increased metabolism of hyperthyroidism, pregnancy, fever, and infectious and toxic states in general, but it falls down completely in regard to the great increase of metabolism with exercise, which increases tolerance and reduces the insulin re-

quirement. Ignorance of these facts has led to suggestions that insulin is needed chiefly during the day when patients are active, and also that bed rest should be used in the severest stages in order to reduce total metabolism. As a matter of fact, there is a strong tendency for the blood sugar to rise while the patient is sleeping during the night, and the greatest increase of insulin requirement occurs when a patient does not burn up surplus calories by exercise but by inactivity allows them to produce obesity.

In general, then, we may temporarily undernourish a patient if he is too heavy or overnourish him if he is emaciated, but the final diet is one which provides total calories sufficient to maintain strength and comfort, with a body weight either normal or possibly five to ten pounds below the average normal. The slightly subnormal weight will sometimes save a patient from taking insulin, or will reduce the required dosage in such manner as to simplify the management. The proportions of protein and carbohydrate are subject to considerable variation according to the needs of the patient and the preference of physicians. Possibly 80 gm. of each per day may be taken as an average standard. If there is kidney or liver disease, or if the patient does not like protein, there may be a reduction to any desired degree, but if the patient craves protein and there is no contraindication, there may be an allowance of 120 gm. per day without harm and without much influence on the insulin requirement. A reduction of carbohydrate to 40 or even 30 gm. per day is feasible when a special effort is made to avoid using insulin. At the other extreme, some patients feel best with possibly 100 to 150 gm. of carbohydrate per day and may receive it with benefit. The detriment of extremely high carbohydrate diets consists not in the comparatively slight increase of insulin which they require, but rather in the more marked and rapid fluctuations of blood sugar, interfering with the smooth uniformity of the sugar level at all times of day, which is the ideal. So long as a patient is kept sugar-free, it is impossible to produce any dangerous acidosis by any proportions of carbohydrate and fat which he can be induced to eat.

The standard, therefore, is not fixed by acidosis but by a physiological balance of foodstuffs within the limits of the total calories allowed. This balance, determined by the comfort and efficiency of the patient, always provides far more than the minimum of theoretical glucose content to prevent acidosis. Finally, it should be emphasized that the ideal treatment can never consist in fixed rules or charts applicable to everybody, but, on the contrary, every patient should be studied as an individual and a diet worked out to suit his individual needs. The fundamental requirement of limiting total calories and body weight as stated above is scarcely questioned today by any competent authority. Within these limitations of total calories, there is much room for the judgment and preferences of individual physicians in choosing the proportions of foodstuffs.

INSULIN TREATMENT

If we proceed now to the second division of treatment, namely, the use of insulin, we find that most of the questions regarding it are answered in advance by a thorough grasp of the principles of diet. There is no possible ratio between the quantity of insulin required and the quantity of carbohydrate in the diet or of sugar in the urine. We give insulin in one, two, three or rarely four injections in the 24 hours, choosing the smallest number of doses which will keep the blood sugar approximately normal throughout the 24 hours. It is unnecessary and illogical to give insulin always with meals, and different timing of doses may be found advantageous in different cases[7].

The complications of diabetes may be mentioned at this point, because they usually create a special need for insulin. This is most marked in coma, which may sometimes demand several hundred units of insulin in 24 hours. The other measures which were used before the discovery of insulin still remain important, the greatest emphasis now being placed upon an abundant supply of fluids, while alkali is used only in moderate dosage. Also, carbohydrate is generally to be administered in the form of fruit drinks or intravenous glucose injections, even though the blood sugar is already high. There can be no doubt that coma has become less frequent and its treatment far more successful since the introduction of insulin.

Gangrene has apparently not diminished in frequency, and the losses of life and limb from it are still high. The treatment is fairly well standardized; still the combination of the best medical and surgical skill very largely determines the outcome and the results in different hospitals vary widely. On the surgical side, every operator is inclined to think that the removal of a gangrenous limb is a simple matter, but the decision when to be conservative and when to be radical, and all the details of operative technique are problems which create an enormous balance in favor of the surgeon with special diabetic experience. On the medical side it must chiefly be emphasized that most cases do best with moderate undernutrition for a number of days or weeks. High diet and high insulin dosage are not equivalent to lower diet and lower insulin in their influence upon healing. In The Physiatric Institute we also make the diet salt-free in all gangrene cases, in order to relieve any existing edema or hypertension and also on general principles for the arteriosclerosis. We consider it to be beneficial not only in the immediate results but also in its influence on the ultimate outcome and longevity.

Much attention is being paid nowadays to the vascular lesions in diabetes, of which gangrene is only one manifestation. Hypotheses of the nature and etiology of all these vascular changes are being discussed, and one of these is the causation by fat-rich diet. It is not established that the atheroma produced in rabbits and other animals by cholesterol feeding is identical with that of human patients. Furthermore, though I have opposed diets with excessively high fat content, I doubt whether they will produce arteriosclerosis in patients who are kept sugar-free. If we drop theories, we have just one outstanding fact to consider, namely, that every patient who has active diabetes for perhaps ten years or longer has demonstrable arteriosclerosis. This rule applies to mild as well as severe cases, and even young patients develop sclerosis of their vessels if they happen to live long enough with uncontrolled diabetes. On the other hand, patients with sugar-free urine and normal blood sugar apparently do not develop arteriosclerosis any more than non-diabetics of similar type. Whether the direct cause consists in the sugar itself, or the faulty nutrition and abnormal metabolism, or in some of the other factors championed by different writers, the clinical lesson seems clear and obvious. Keep your patients under proper control and they will be safe from vascular lesions as well as from other dangers. Some persons may inquire why we insist on maintaining normal chemistry of urine and blood as strictly as may be possible, and some physicians, including some specialists, have heretofore considered it permissible for patients to have excessive sugar, at least during some portion of the day, as a safeguard against hypoglycemia and as an escape from the inconvenience of precise diet. It may be answered that glycosuria and high blood sugar are decidedly abnormal conditions, and nobody can expect to go on with such abnormalities for months and years without suffering some kind of harm. Therefore, even if coma and other acute dangers are prevented and the patient is kept in apparent health and comfort, careless treatment may still be disastrous in the ultimate outcome.

The general results of lax management are also apparent without waiting so long. Diabetes has been scientifically mastered. Theoretically, every patient can be expected to live out his full natural lifetime, limited only by such permanent lesions as have already developed. Statistically, there has been no fall in the death rate. This situation seems to be unparalleled in any other department of medicine in which revolutionary advances in treatment have been made. Some writers analyze the figures as indicating a small drop in the death rate, on the ground that more cases are constantly being diagnosed and also the death certificate regularly mentions diabetes even when a death occurs from old age or any other cause. The differences, nevertheless, are trivial in proportion to the transformation that could have been accomplished before now if the available means of treatment had been efficiently utilized. We must look for the chief cause of the continued high death rate in the human factor; in other words, it is the fault either of the patients or of the doctors. Disobedience of patients is undoubtedly one large element. The remissness on the part of the medical profession may be variously appraised according to individual opinion. A surgical operation is dramatic and impresses a patient as important, and he is, therefore, inclined to seek the best surgeon available and follow his advice unreservedly. On the other hand, the patient with a metabolic disorder is apt to refer to his treatment as "nothing but a diet," and he is inclined to be more indiscriminate in his choice of adviser and more reckless in doing as he pleases. The value of institutional care for starting treatment and especially for training the patient is generally recognized. Granting that specialists in every department are disposed to stress the importance of their own work, and making allowance for any prejudice thus involved, I would still hazard the following statement. If all skilled surgeons in this country were suddenly eliminated and all operations were to be performed by physicians with no previous special training in surgery, there would be a rise in the surgical death rate, but this rise would be insignificant in comparison with the enormous number of present-day diabetic deaths which could be prevented by sufficient skill on the part of the attending physician. The general medical practitioners are the ones who see the overwhelming majority of these cases, and the reduction of the mortality is a problem to be solved by them through the medium of their instructions as regards treatment and by training patients to heed instructions.

REFERENCES

1 Allen, F. M.: The rôle of fat in diabetes. New York M. J. 104:1005-1007, 1916.
2 Allen, F. M., Stillman, E. and Fitz, R.: Total dietary restriction in the treatment of diabetes. Monograph No. 11, Rockefeller Institute for Medical Research, 1919.
3 Allen, F. M.: Experimental studies in diabetes. Series I. Production and control of diabetes in the dog. 4. Control of experimental diabetes by fasting and total dietary restriction. J. Exper. Med. 31:575, May 1920.
4 Allen, F. M. and Wishart, M. B.: Alcohol in the diabetic diet. J. Metabolic Research 1:281, Feb. 1922.
5 Sherrill, J. W.: Clinical observations with insulin. 2. The influence of carbohydrate and protein on diabetes and the insulin requirement. J. Metabolic Research 3:13-61, Jan. 1923.
6 Allen, F. M.: Clinical observations with insulin. 3. The influence of fat and total calories on diabetes and the insulin requirement. J. Metabolic Research 3:61-177, Jan. 1923.
7 Allen, F. M.: Timing of insulin doses. J. A. M. A. 82:1937, June 14, 1924; also, J. M. Soc. New Jersey 21:223-224, July 1924.

Discussion

Dr. Harry T. French, Hanover: First I want to thank Dr. Allen for talking to us.

I will present a case that brings out some of the points which Dr. Allen made this afternoon.

A woman, 22 years of age, who has been diabetic for three years, was brought to the hospital in March, 1928, in diabetic coma.

The first time she had been seen by a physician for diabetes was in her home. She was in coma then and was given large doses of insulin. She was nearly out of the comatose condition when she entered the hospital.

The blood sugar at that time was 122 milligrams per 100 c.c., and the blood urea was 42 milligrams per 100 c.c. Under treatment of a dietary character her condition cleared up rapidly. The sugar disappeared, and the acetone and diacetic acid cleared from the urine. At this time she had a low kidney threshold for sugar, showing sugar in the urine, with a blood sugar of 67 milligrams per 100 c.c. She was discharged from the hospital after she had been sugar-free for a week, on a diet with 24 units of insulin three times a day. She continued to follow that diet under supervision until August of that same year, when, like so many diabetics, she visited relatives for a week, dropping the insulin and every safeguard in the diet because she felt so well. At the end of the week she went into a deep coma, and was brought to the hospital. The blood sugar at this time was 333 milligrams per 100 c.c., with large quantities of acetone and diacetic acid, together with albumin and acetone in the urine.

A week later the urine was sugar-free, there was no acetone or diacetic acid, but the blood sugar was 326 milligrams per 100 c.c.; that is, the urine was sugar-free but the blood sugar was about twice the normal. The normal level of blood sugar at which sugar appears in the urine is between 160 and 180 milligrams per 100 c.c., but she had 326 milligrams and was not showing sugar in the urine at that time.

Going back to the previous admission, she had a low kidney threshold, showing sugar in the urine with a blood sugar of 67 milligrams per 100 c.c. of blood. There had been a definite shift in the kidney threshold between these two admissions. She developed an insulin reaction about a week later with a blood sugar of 58 milligrams per 100 c.c. on a dosage of 32 units of insulin three times a day. Shortly after this she was sent home on 16 units three times a day and continued sugar-free until about a month ago.

About a month ago, an acquaintance told her she she could take pills by mouth instead of insulin injections, and she did this for about a week, neglecting her diet at the same time, and again she dropped into coma and was brought to the hospital. The blood sugar was 500 milligrams per 100 c.c. with a strong acetone reaction. We could not get any urine at this time. Later, urine was obtained; it was filled with sugar, acetone, diacetic acid and albumin. The stomach was washed out at this time. It was filled with food that had been taken 36 hours before. The patient was given 150 units of insulin in divided doses with intravenous glucose, and in 30 hours she regained consciousness. She gradually improved and was not showing sugar in the urine but in spite of this developed an insulin reaction; the blood sugar at this time was 60 mg. per 100 c.c.; that is, the kidney threshold was low again, and the blood sugar varied from 500 milligrams per 100 c.c. to 60 milligrams per 100 c.c. during the following 12 hours.

I have a slide that illustrates the curve of the blood sugar and sugar output with the time relations.

[Slide shown.]

Because of this instability she was placed on small feedings six times a day with small doses of insulin with each feeding. A urinary sugar curve was plotted with hourly quantitative determinations. This shows that in 24 hours she lost about 60 grams of sugar out of an available 130, in spite of being sugar-free all day. That is, this curve of the urinary sugar starts off with the base line here [pointing], at 7 o'clock in the morning with the urinary sugar at 12.6 grams; in the next hour it was 13; it dropped to 4.6 in the next hour and then .2, so that at 11 o'clock the urine was sugar-free and ran sugar-free until 7 o'clock in the evening, when sugar began to show again in the urine, and at 10 o'clock the urine analysis showed 3 and a fraction grams; the next specimen showed about the same amount, and then during the night period until 5 o'clock in the morning no urine was obtained. During that whole period she passed only 3.7 grams of sugar, but beginning at 5 o'clock until 8 o'clock, that is, a period of 4 hours, she passed 23 grams of sugar. That is, at this time, for some reason the blood sugar increased very markedly, and there was a flooding of the circulation with sugar.

Now, to go back to the curve, she had a feeding at 7 o'clock, another at 9, one at noon, another at 2, one at 3.30, and another one at 7, with insulin with each of these feedings, and she ran along sugar-free. During the night she had no food and no insulin, and she continued through the night period until she began to throw out large quantities of sugar. She was given a dose of insulin at 7 o'clock and another at midnight, and she became sugar-free throughout the 24 hours.

This case brings out certain features that are rather interesting to me. One is the fact that the level at which sugar is spilled will vary in a kidney, depending upon other factors. It may vary from a low level. In this case the patient showed sugar in the urine when the blood sugar was 67 milligrams per 100 c.c., and she had shifted from blood sugar of 476 at this point; that is, there had been a very rapid drop, but at the other end of the curve there was marked flooding. This flooding during the night, of which Dr. Allen spoke, is very suggestive of the overflow of sugar that you get in animals with experimental puncture in the region of the third ventricle, and it is questionable whether, in an unstable condition, it is not a nervous phenomenon that brings out sugar in this way as the insulin is used up. It also shows that a graphic representation of the patient's condition may aid in determining the advisable method of treatment.

Dr. C. O. Coburn, Manchester: I want to thank Dr. Allen for bringing up the point that there is no ratio between insulin and carbohydrate. That is something which has troubled me a lot. In the past I have tried to give insulin in proportion to the amount of carbohydrate and it has not worked. In some cases I could give a little and get results, and in other cases it would take large quantities.

In the treatment of diabetes from the general practitioner's standpoint, I think it is necessary to make the treatment as simple and as understandable as possible to the patients and at the same time to give them the impression how serious a matter it is and the absolute necessity of keeping sugar-free, as Dr. Allen has mentioned. On most of my patients, or a good part of them, I do not use insulin if I can keep them on such a diet as will maintain the body weight where it ought to be, generally a little below normal,

and allow them to do their usual work. If no sugar is present, I am fairly well satisfied. They are all told to check up on themselves every day, and at the first indication of sugar they are supposed to report. In cases where insulin is indicated, they must go to a hospital. This is my personal view of the matter. I think it is precarious to start a patient off without a lot of instructions. I think they should go to a hospital and be under direct supervision; not only that, but it gives the patient the impression that this is an important thing, it is something which has to be followed, and my experience has been that if you take the patient to the hospital and teach him there how to use insulin, how to examine his urine, and how to make up his diet, it will result much better than office treatment.

At present I am not using absolute weighed diets in most of my cases. I am using measured diets or approximate diets, and watch the weight and the urine. I have obtained very good results in the mild cases. In serious cases I insist upon the diet being weighed and followed minutely.

There is one point I should like to suggest, that in all cases of surgery, infectious diseases or any condition where a diabetic is running a temperature, he should receive very intensive treatment and if the doctor is not familiar with the use of insulin, he should have some method whereby the patient may receive intensive insulin treatment against hyperglycemia.

I want to thank Dr. Allen again for his very fine address.

PYURIA IN CHILDREN*

BY RICHARD W. ROBINSON, M.D., LACONIA†

BECAUSE of the common occurrence of a pus-containing urine in childhood; because often this condition, particularly in young children, goes unrecognized for the reason that routine urine examinations, in the first few years of life, are by no means universal; and principally because pyuria from a therapeutic standpoint is not a clinical entity, but rather a symptom accompanying several, varying, pathological conditions of the urinary tract, there would seem to be sufficient excuse for taking again an already well-worn trail, and some slight extenuation for the type of paper this must be owing to the necessities of a limited personal experience.

My own interest in this subject was freshly aroused by Dr. John D. Lyttle of New York, who presented a paper of the same title before the Connecticut Clinical Conference last September; the conclusions that could rightly be drawn from his material were such that they gave an entirely new emphasis to certain phases of the less common methods of therapeutic approach. This paper was not published but was abstracted in the November issue of the *Yale Journal of Biology and Medicine*. The thesis presented was, in brief, that all cases of acute or recurrent pyelitis, not dependent upon structural abnormalities of the urinary tract, are in reality expressions of a focal interstitial nephritis, cortical or medullary, or both, which, in turn, owes its origin to blood-borne organisms from some primary focus and which produce, either by direct extension or sowing, the usually meager pathological changes found in the renal pelvis and lower urinary tract. The conclusions were obvious and logical. As these local nephritides tend to heal in from eight days to three weeks, and as the known urinary antiseptics cannot act upon the diseased and functionless areas in the kidney, the present accepted methods of treating these cases are, in reality, unnecessary and non-effectual administrations of an often expensive preparation. The only real merit of the procedure, therefore, depends upon the additional quantity of fluid that is urged upon the patient during the period of treatment. The logical corollary deduced was that rational therapy demands a concentration of attack upon the primary focus, be it of acute infectious or chronic infectious nature.

The importance of knowing whether these conclusions constitute the whole truth, or only a part of the truth, is self-evident and from an effort to determine just where the weight of the available proofs rests, the material to be presented in this paper was gathered.

A review of this subject in such current textbooks as Scherer[1], Morse[2], Lucas[3], Holt[4], Abt[5] brought conviction that either beliefs have become stereotyped to the state of dogmas or that there should be sufficient evidence in the literature to contradict any approach to universality of the hematogenous nephritis concept. The textbook descriptions of pyuria in children are so nearly alike that it makes no important difference which one is used as a reference. For definition of the subject under discussion, I can, perhaps, do no better than quote in part from Helmholz' chapter in Abt's *System of Pediatrics:*

"Pyogenic infections of the urinary passages are manifested by the presence of bacteria and pus in the urine. The infection is usually caused by a member of the colon group of organisms and clinically it is marked by fever, pallor, and the absence of other clinical symptoms.

*Read at the Annual Meeting of the New Hampshire Medical Society, at Concord, May 13, 1930.

†Robinson—For record and address of author see "This Week's Issue", page 1171.

"Except for common colds and gastro-intestinal disturbances of infancy, pyelitis is one of the commonest diseases of childhood. In eight years of experience, Göppert found that pyelitis made up about 1.2 per cent. of his entire practice. Smith estimated that it comprises about 1 per cent. of all cases seen by pediatricians.

"Pyelitis has been rightly termed as a disease of the diaper age, for beyond the second year it decreases rapidly in frequency. In older children it is usually a more serious disease, although not nearly so common. It is most often encountered during the second and third quarters of the first year, and is rather rare in the first six months, although there is a very definite type which occurs in the first ten days of life, as described by Smith, Kovalesky and Moro, and Helmholz.

"Escherich was the first to point out the great prevalence of pyelitis in girls, and various observers have described from 57 to 100 per cent. of cases in girls.

"There are two definite periods of the year when pyelitis seems to be more common; in the summer, when it is associated with gastro-intestinal disturbances, and in the winter, when it follows infections of the upper respiratory tract. The infections, whether during summer or winter, are in most instances due to the colon bacillus.

"Predisposing factors must be considered under two headings: (1) pyelitis which is primary in infants and children, and (2) pyelitis, which complicates a great variety of ailments, affecting practically any of the other systems of the body. It must be assumed that retention is the most important factor in infection. Chilling, exposure on auto rides, sitting on cold floors, or in drafts, are repeatedly given as the possible exciting causes.

"It should be noted at the outset that the usual mode of infection in pyelocystitis has not been definitely determined. Furthermore, the primary seat of infection is not yet known, and in only relatively few cases has the location of the inflammatory process in the urinary tract been ascertained.

"The evidence thus far presented indicates that pyelitis may be caused either by the ascending, or the hematogenous route. Knowledge, with regard to which infections are limited to the bladder, and which involve the entire urinary tract, is still so fragmentary that it is inadvisable to attempt to decide which is the more likely mode of infection.

"Numerous observers have noted the frequency of relapse in pyelitis. Göppert recounts several instances of recurrence, even after years of apparently normal health and negative findings in the urine. Ross, on the other hand, contends that these cases are reinfections rather than relapses. In almost all articles on pyelitis the frequency of relapse is commented on.

"The disease in the infant, whether severe or mild, is characterized by the absence of any signs localizing the condition in the urinary tract. Even in severe cases, bladder symptoms are present in less than 3 per cent.

"The course of pyelitis in patients with normal urinary tracts is usually favorable. The acute symptoms may be very severe and the temperature may reach 106 degrees and remain elevated for days. It is extremely rare, however, that infants die from simple acute pyelitis. Almost invariably it is the complicating nephritis which causes death.

"As a rule, the acute general symptoms, fever, restlessness, and lack of appetite, persist for ten days or two weeks. In the severe cases an acute course may last from three to four weeks in spite of all therapeutic measures. Exceptional cases continue for years."

From the above description certain features attract the attention as possessing a controversial importance to the questions at issue: (1) that there are two definite but not necessarily unrelated groups of cases, one group representing patients with abnormalities of the urinary tract leading to retention, and one group in which no such abnormalities are present; (2) the apparently greater preponderance of cases among females; (3) the seasonal incidence, coinciding with the seasonal incidence of the upper respiratory and gastro-intestinal disturbances; (4) the usual duration of ten days or two weeks in the favorable cases and the frequently obstinate course of those cases that run over this period; (5) the tendency to relapse or recurrence.

The importance of the obstructive pathologies and abnormalities of the urinary tract as contributory factors to the incidence and continuance of urinary infection, has been stressed by Helmholz[6], Bugbee[7], Campbell[8], Brewer[9] and Hunner[10], to mention but a few. It is a demonstrated fact that urinary stasis is commonly superseded by infection and it would seem probable that the infecting organism finds its way down from above, rather than up from below the point of retention. It appears probable, likewise, that in this group of cases a temporary improvement, even to the point of apparent cure, so far as evidence of infection is concerned, might be obtained from the use of truly potent urinary antiseptics, but it is obvious that this improvement is subject to almost certain relapse as soon as the effect of the antiseptic is stopped and conditions recombine to bring about a new infection in the retention area. If the condition of retention plus infection is permitted to persist, it almost always leads to destruction, complete or partial, of one or both kidneys, depending upon the ability of the retention to exert back pressure on one or both sides.

The pathological changes described in such kidneys are classified usually as pyonephrosis or

pyelonephritis and are quite different from the type of interstitial nephritis described by Lyttle, Chown[11] and Schloss[12], in their articles of the past two years. Reason demands that this group of cases be diagnosed and treated from the viewpoint of relieving the obstruction, if unnecessarily grave consequences are to be avoided.

The group of cases without a retention basis, occurring in children, is the most interesting to the general practitioner and is described as constituting far the larger number. How correct the observations on these have been I do not know, since a consideration of many of the reported cases does not remove all doubt in regard to the accuracy of the diagnosis, especially in many of the female infants. Be this as it may, it is around this group that most of the controversy regarding etiology and therapy wages, and about this group that most of the recent literature dealing with question of pyuria in children has been written. These are the cases of so-called acute pyelitis, although when one attempts to find substantiation for this nomenclature there is but very little in the way of described pathology to uphold it. The most complete descriptions dealing with the subject are those of Helmholz[13], working with experimental infections of the urinary tract in rabbits, and of Chown[11], and Wilson and Schloss[12], from autopsy and cystoscopy material. Helmholz demonstrated that, with the particular organism he was using, infections of the unobstructed urinary tract could be induced in rabbits either by the hematogenous or the ascending route and he found that the pathological lesions produced by the two modes of infection differed pronouncedly, one from the other. The blood-borne infections expressed themselves as cortical, or medullary abscesses, or both, while the ascending infections expressed themselves by inflammatory lesions of the parietal wall of the renal pelvis. Chown, in reporting thirty autopsies, concludes that the term *pyelitis of infancy* is a misnomer, as the usual microscopic pathology consists in an interstitial nephritis. Wilson and Schloss reach the same conclusions from a series of forty-nine autopsies.

I was not able to find in the literature any real evidence upholding the theory of ascending infection, save the above mentioned articles of Helmholz and the oft-repeated argument that, as the disease is so much more common in girls than in boys, the apparent ease with which the affecting germ could find access to the female urinary tract was the probable explanation of the phenomenon, the infection being a direct ascending one or an ascending one by way of the lymphatics.

To measure the weight of this argument let us consider the facts. Graham[14] reports six cases in newborn infants, three girls and three boys; Lord[15] reported 50 cases, 75 per cent. of which occurred in females, but he does not leave his cases clear of diagnostic doubt. In the cases reported by Chown, Wilson and Schloss, 150 in number, the incidence was about 60 per cent. in girls and 40 per cent. in boys. They explain this by presuming that the plus 20 per cent. occurrence in females is probably due to ascending infection, although their pathological studies show no greater evidence of this in girls than in boys.

Letting this rest for the time being, consider the probable import of the seasonal fluctuations of these urinary infections as described by Helmholz[13], Gannon[16] and others. The peak in March accompanies the peak of the upper respiratory infections, and the peak in July-August accompanies the peak of gastrointestinal disturbances, with the undoubtedly frequent association of pyuria as a complication or aftermath of these infection processes. Consider the evidence presented by Schwartz[17], who demonstrated that the catheterized urine of children suffering from gastrointestinal disturbances contained colon bacilli in over 60 per cent. of the cases; and of Cabot and Crabtree[18], who showed, in adults, positive blood cultures preceding urinary infection with the colon bacillus. Consider that an acute vaginitis, with a purulent discharge, is a frequent complication of both respiratory and gastrointestinal disease in female infants. Remember that we have all been taught that pyelitis is a disease of the female infant and therefore that we are apt to look for it with more diligence, and accept it with less evidence in the female than in the male. We have, then, on the one hand, further evidence supporting the hematogenous route of infection and, on the other, a plausible explanation of the apparent higher incidence of pyuria in the female. It, therefore, seems to me that the weight of the available proofs are at present on the side of hematogenous nephritis as the common initial lesion of this case-group.

Having arrived at these tentative conclusions, how does reason prompt us to deal with the individual patient for whom it may fall to our lot to care? First, the diagnosis must be made, and this can be made in no other way save by microscopic examination of the urine. I cannot urge too strongly that this be done routinely and frequently, if we are to live up to those diagnostic ideals which, I think, to most of us provide the chief inner satisfactions of the practice of medicine. To obtain urine for examination from an infant or child is not so easy as in the case of adults and yet it is too simple a procedure to have its difficulties afford any excuse for not doing it. In infants of the diaper age, a test tube attached with adhesive strips in the male, and a clean rubber glove or small spica urinal in the female, will provide a ready means of accomplishment. The urine, once obtained, should be well shaken and, without centrifuging, a drop should be examined under the low-power

microscope. The presence of more than six to eight pus cells to the field is definitely suspicious of pyuria, but vaginitis and inflammations under a tight foreskin must be ruled out. In females this is done only by catheterization; in males by carefully washing the glans and sulcus, or, where this is impossible, by catheterizing. If in this specimen the pus content is still more than six to eight cells to the low-power field in an uncentrifuged preparation, the diagnosis of pyuria is made. I am purposely leaving out a discussion of the bacteriological diagnosis, as at present there has been considerable doubt thrown up on its value, and it is certainly not available to most of us.

Once diagnosed, a reasonable procedure is to treat each case as if it belonged to the non-retention group, concentrating upon the treatment of any acute infectious process, if present, and if not, removing so far as possible chronic foci of infection. The forcing of fluids and the feeding of alkalies to the extent of rendering the urine alkaline are unquestionably beneficial.

If, after four weeks, the condition is not markedly improved and there is no obvious source of infection, the case is, in all probability, one of the retention group and should be turned over to a competent specialist for a thorough urological examination.

It is my impression that urinary antiseptics should be withheld until this four week period is up, as the improvement produced in retention cases might well be the cause of disastrous procrastination. I would make one exception to this rule and that is in those rare cases in children whose illness is ushered in by bladder symptoms, for here, as in the adult, we may be dealing with a cystitis and here, as well as in the adult, antispasmodics and antiseptics should afford relief.

REFERENCES

1 Scherer: Diagnosis of Children's Diseases.
2 Morse, J. L.: Clinical Pediatrics. W. B. Saunders Co., Phila., 1926.
3 Lucas, W. P.: The Modern Practice of Pediatrics. Macmillan Co., N. Y., 1927.
4 Holt, L. E. and Howland, J.: Diseases of Infancy and Childhood. 9th ed., D. Appleton & Co., N. Y., 1926.
5 Abt, I. A.: Pediatrics. W. B. Saunders Co., Phila., 1923-26.
6 Helmholz, H. F.: Congenital abnormalities of urinary tract in childhood. J. A. M. A. 89: 1932, Dec. 3, 1927.
7 Bugbee, H. G. and Wollstein, M.: Surgical pathology of urinary tract in infants. J. A. M. A. 83: 1887, Dec. 13, 1924.
8 Campbell, M. F. and Lyttle, J. D.: Ureteral obstruction in infancy; study of 74 cases. J. A. M. A. 92: 544, Feb. 16, 1929.
9 Brewer, G. E.: The present state of our knowledge of acute renal infections. J. A. M. A. 57: 179, July 1911.
10 Hunner, G. L.: Ureteral stricture and chronic pyelitis in children. Am. J. Dis. Child. 34: 603, Oct. 1927.
11 Chown, Bruce: Pyelitis in infancy; pathological study. Arch. Dis. Child. 2: 97-118, April 1927.
12 Wilson, J. R. and Schloss, O. M.: Pathology of so-called "acute pyelitis" in infants. Am. J. Dis. Child. 38: 227-240, Aug. 1929.
13 Helmholz, H. F.: Pathologic changes in experimental, ascending and hematogenous pyelitis. J. Urol. 8: 301-306, Oct. 1922.
14 Graham, R. H.: Six cases of pyelitis in newly-born infant. Am. J. Sci. 170:401-404, Sept. 1925.
15 Lord, R. M.: Fifty cases of pyelitis in children. Rhode Island M. J. 9: 51-54, April 1926.
16 Gannon, N. D.: Etiology, diagnosis, and medical treatment of acute pyelitis in children. Atlantic M. J. 31: 456-458, April 1928.
17 Schwartz, A. B.: The bacteriology of the urine in children with vulvovaginitis. Am. J. Dis. Child. 13: 420-425, 1917.
18 Cabot, H. and Crabtree, E. G.: Nontuberculous renal infections. Surg., Gynec., Obst. 23: 495, Nov. 1916.

Discussion

Dr. Frank N. Rogers, Manchester: This subject of pyuria in children is well worth our consideration and has been splendidly handled by Dr. Robinson. He has left very little to be discussed from the standpoint of the pediatrist. The subject of pyuria in children is of interest to everyone practicing medicine. Although theories are many, the etiology of the symptoms or conditions remains one of our medical mysteries. Probably no one theory will account for all cases of pyuria. Our greatest effort lies in the making of diagnosis. It is true that most of us fail to examine the urine from very young children as often as we should, due to the difficulty of obtaining specimens. After the diagnosis of pyuria is made, the patient should be put to bed and treated as outlined in the speaker's paper.

The giving of extensive urinary antiseptics in the general run of cases is useless, if not harmful, in the very early stages of the disease. Protracted cases should be turned over to the urologists, for they are the only ones who are equipped to handle them.

I want to thank Dr. Robinson for this splendid symposium which is the result of a great deal of study and reading, coupled with his own experience.

Dr. Elmer J. Brown, Manchester: I agree with Dr. Robinson and Dr. Rogers that we do not examine the urine of children so often as we should and I think it should always be examined early.

As to the infecting organism, I think that there are more cases of infection from colon bacillus than from any other organism, and in those cases, as Dr. Robinson has suggested, alkalinizing will do more toward killing the bacteria than antiseptics will.

Abnormalities do occur, although I think very few are found because such patients do not, as a rule, reach the urologist—I mean, they are not turned over to him. They go along and get better and eventually may get well, but a great many of them, if investigated earlier, would get well quicker.

Dr. Bugbee and his associates in 4,090 children found 117 with abnormalities which they thought were sufficient to cause great influence in any infection that might exist. These, of course, were all kinds of cases, not necessarily urological cases. Losley and Butterfield found 29 out of 100 cases that showed abnormalities which they believed would cause an influence in infecting the genito-urinary tract. These were, necessarily, urological cases that were sent to them.

There are many abnormalities that can exist; for instance, strictures in infants, beginning rather early. Stricture of the ureter has been found in the fetus by at least two different men. If persistent, I think they should be investigated thoroughly by pyelogram, and so forth.

At the Balch Hospital, this spring, we had an interesting case of pyuria. This was a child nine years old, a girl, who was admitted on January 8, 1930. The complaint was painful urination. The physical examination showed nothing. On examination of the urine it was found to be alkaline, and contained a large amount of albumin and a large amount of pus. A tuberculin test was positive on January 9. On January 27 the urine was still cloudy, and on February 15, on cystoscopy of the patient, the bladder was found filled with pussy urine. I used a 24 cystoscope, because I wanted to be able to use a large catheter through it. We dilated the ureter on the right side where there was evident stricture and put in a catheter. The catheter was left in for about two days. Argyrol was put in several times during that time, and the patient was discharged.

That was February 15, and on March 28 the child was very much better. The temperature was normal, and the urine we took for guinea-pig inoculation on the 15th came back, April 2, negative for tuberculosis. I thought at first this was a tuberculous kidney, but from the fact that it came back negative I think there must have been some other infection. However, the patient cleared up very well and has gone home.

DR. R. W. ROBINSON: I have nothing further to add to my paper, except in regard to the procedure mentioned by Dr. Brown—that is the effect of alkalinizing the urine in these colon bacillus cases. It seems to be a very effectual procedure, much more effectual, for example, than the use of antiseptics, but why, apparently nobody knows. As to the chemistry involved,—the actual degree of alkalinization that is brought about,—the PH. value of the urine is way below that degree of alkalinity necessary to produce any detrimental effect upon the colon bacillus in the test tube.

As to the bacteriology of this condition in general: Dr. Lyttle reported quite a series of cases —it is too bad his paper was not published—in which there were recovered the same organisms in the urine that were found in focal infections of the teeth and tonsils. He thinks the infection of the urinary tract due to blood-borne organisms. Along that line there has been a good deal of work done recently on the presumably normal blood and urinary tract, a large percentage of which, when watched for any great period of time, shows positive blood cultures and positive urine cultures without other evidence of an infectious process. So the bacteriology of the condition, as I said before, is undetermined.

THE PRIZE ESSAY*

THE PREVENTION OF DISEASE

Some Applications of Preventive Medicine

BY EDWARD O. OTIS, M.D., EXETER†

PRESIDENT ELIOT once remarked, with his prophetic vision, that the physician of the future would be largely engaged in preventive medicine. While this Utopian era has not yet arrived and probably never will, yet there has never been so much activity in the prevention of disease as at the present time. One has only to consider the ever broadening scope of official health departments—National, State, and Local —and the almost innumerable voluntary health organizations to realize this fact.

WHAT OFFICIAL HEALTH DEPARTMENTS DO

Beside the supervision of the water supply, the sewerage, the milk supply, and the control of epidemic diseases, state and city health departments have assumed many other duties for the protection of their citizens against preventable diseases.

In their laboratories they make free examinations for the detection of the diphtheria bacillus; the sputum for tuberculosis; the blood for the Widal test in typhoid fever; the Wassermann test for syphilis; and for the determination of the type in pneumonia. Food and drink products are tested as to their purity. These public laboratories furnish free toxin-antitoxin and antitoxin for diphtheria; and antitoxin for immunization against typhoid fever; and vaccine for protection against smallpox.

A large amount of literature is supplied by these official health authorities to the physicians and laity regarding the prevention of disease; the early diagnosis of tuberculosis and cancer; and in some states public clinics are held for the detection of these diseases. Indeed, the literature sent out by official health authorities at irregular and periodic intervals would form a good-sized book on the most modern methods of preventing disease.

*The Trustees of the New Hampshire Medical Society awarded the prize, one hundred dollars, to Dr. Edward O. Otis, Exeter, for the most worthy essay submitted to them.

†Otis—Honorary Director, National Tuberculosis Association. For record and address of author see "This Week's Issue", page 1171.

VOLUNTARY HEALTH ASSOCIATIONS

Not only are these multifarious measures for the prevention of disease carried on by public health officials, but many voluntary unofficial health associations exist in this country for the same purpose. There is the great Rockefeller Foundation which, since its foundation in 1913, has expended the vast sum of $144,189,000, and in 1928, $21,690,738 for the development of medical science and the prevention of disease throughout the world. Through its efforts yellow fever, hookworm disease, and malaria have been almost banished or greatly reduced. Its health touch is felt in almost every country in the world.

Another notable voluntary health agency is that of the National Tuberculosis Association, founded twenty-five years ago, with an annual income of nearly $300,000 mostly derived from a five per cent. tax upon the receipts of the Christmas Seal Sale in the various states. Besides its many other activities in the prevention of tuberculosis, one of its noteworthy undertakings is it research work carried on by able and well-known scientists with the ultimate object of discovering means for the cure and prevention of tuberculosis.

The present nation-wide propaganda for the early diagnosis of tuberculosis at a time when it can be most successfully treated is sponsored by this association.

Then there are the Cancer, Heart, and Mental Hygiene Associations, and others all bent on saving life and preventing disease.

PREVENTIVE WORK OF LIFE INSURANCE COMPANIES

Very valuable work in disease prevention is now being done in this country by some of the great life insurance companies; notably The Metropolitan Life Insurance Company of New York.

In the last fifteen years it has expended a total of $18,709,000 in the conduct of its public health campaign for industrial policyholders and others.—(Dublin.) This same company appropriated $200,000 for the Framingham Demonstration, and during the years of its existence, the death rate from tuberculosis in Framingham has declined from 121 per 100,000 for the decade preceding 1917 to 38 per 100,000 in 1923.

For many years this company has widely disseminated literature of an authoritative character on matters connected with health preservation. It offers periodic examinations to its insured and officials. In its health advertising, by its abundant and attractive free literature, this company furnishes an excellent example of how to "sell" health, for as Professor Rosenau says: "The greatest difficulty in teaching preventive medicine is getting to the people."

MANUFACTURING AND COMMERCIAL COMPANIES

Large manufacturing companies now, quite generally, employ full-time physicians, a large part of whose duties is to see that the employees are kept well, and are in good health when they enter the industry. Some go so far, as the Dennison Manufacturing Company, to require the heads of departments to take a certain amount of exercise in order to keep in condition.

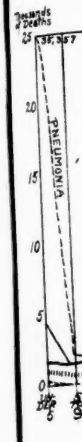
REDUCTION OF DISEASE

If one studies the general mortality statistics for the past two decades, he will observe that in a number of diseases the mortality has been greatly reduced. For example: In New Hampshire, for 1928, the mortality from diphtheria was 5.7 per 100,000; from typhoid fever 2.2; from tuberculosis (all forms) 67.3, and from diseases of early infancy 87.5. On the other hand, certain diseases have increased. For example: In the same state of New Hampshire, the deaths in 1928 from heart disease were 300.9 per 100,000, and from cancer 140.4.

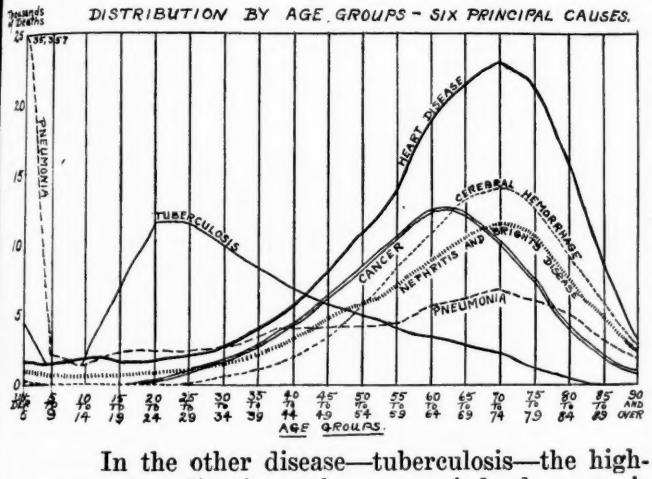
In general, however, the mortality from disease throughout the civilized world has decreased, and the average expectancy of life from birth in 1926 was 57.74 years while a century ago it was 33 years, and down to the discovery of America it was only 18. Such has been the progress of preventive medicine. In reading the literature of early ages, one is struck with the early deaths of so many of the actors in the drama of those times when no knowledge of preventive medicine or sanitation existed. Take for example the history of the illustrious family of the Medici of Italy. A family, which, from their abundant wealth, were enabled to surround themselves with the best their civilization afforded, and yet it is striking to see how few reach old age—many of them dying at an early age of infectious diseases. At this stage of the world's progress, it is difficult to conceive of the enormous mortality which occurred at that epoch from infectious diseases such as the plague, smallpox, typhus fever, epidemic dysentery, leprosy, cholera, and others, all preventable diseases. In the great plague epidemic in London in 1665, 70,000 inhabitants perished. "When men everywhere began to despair, every heart failed from fear, people were made desperate through the anguish of their souls, and the terror of death sat in the countenances of the people." (Defoe.)

If one will look on the chart of the so-called six "capital" diseases, namely: Heart Disease, Cancer, Nephritis, Cerebral Hemorrhage, Tuberculosis and Pneumonia, he will observe that the curve of mortality varies in each disease at different age periods.

The mortality at certain age periods is far higher than at other age periods. In pneumonia, for example, the high points in the mortality curve are under five years of age and from sixty-five to seventy-five years of age, while in tuberculosis the high points are under five years of age (in infancy) and from twenty to thirty-five years of age. The economic significance of



this is apparent and striking. In the former disease—pneumonia—the greatest mortality is in infancy where there is only a possibility of future usefulness, and in the later years of life when a man's life work is practically accomplished.



In the other disease—tuberculosis—the highest mortality is at the age period when one is entering and in full swing of his life's work—the most economically valuable period of life.

I now propose to consider some of the predisposing causes and their prevention of these high peaks in mortality in tuberculosis, heart disease, pneumonia, and some of the others of lesser mortality.

TUBERCULOSIS

Infants

In the registration area of the United States, the mortality from tuberculosis under one year of age in 1926 was 64.6 per 100,000 while under five years of age it was 39.6, and between five and nine years of age it was only 11.9.

What is the cause of this comparatively high mortality from tuberculosis in infants? The infection must come from near at hand as the infant cannot move about—from a tuberculous mother, or some member of the household, as a grandparent or old nurse with a chronic cough which may indicate a latent tuberculosis. When infected, which easily occurs, the infant almost invariably dies for it has not developed any resistance. There are two ways of avoiding this calamitous result. First the immediate removal of the infant from its infected environment, according to the well-known plan of Professor Grancher of Paris, who removed such infants to healthy homes in the country. It has been found that when calves have been removed from their tuberculous mothers, they grew up healthy animals. The second way is to treat the infant, within ten days of its birth, with the so-called B. C. G. (Bacillus Calmette-Guerin) vaccine now greatly in vogue in France and in other European countries. (See *New England Journal of Medicine,* Oct. 31, 1929—Vol. 201, No. 18, p. 903.)

THE B. C. G. VACCINE

This is an avirulent live bovine bacillus grown on a medium of glycerated potato saturated with ox bile. After a series of cultivations extending over a considerable series of years, an avirulent live tubercle bacillus was produced which after many trials with laboratory and other animals, was found not to produce tuberculous disease, but to prevent it, when virulent tubercle bacilli were introduced in the animal. Thousands of infants in France, in tuberculous families, have been treated with this vaccine and the reports have been uniformly favorable. Many statistics are already at hand, mostly French, and perhaps should be taken with some allowance on account of Gallic enthusiasm, showing a striking diminution of infant mortality from tuberculosis, in infected families, in vaccinated infants.

Calmette and Plotz (*American Review of Tuberculosis,* June 1929, Vol. 19, No. 6, p. 567) give the following results obtained from 2,368 vaccinated infants and 4,854 non-vaccinated. The mortality from all causes in the non-vaccinated children (up to four years) was 21.4 per cent., of which 15.9 per cent. were tuberculous, while the mortality from all causes among the vaccinated children was only 11 per cent., of which 3.4 per cent. were tuberculous. Calmette and Plotz further state that B. C. G. has been given to 132,000 infants from July, 1924 to January, 1929, and in no case has a deleterious effect followed. The same results were obtained in Roumania where, in the city of Bucharest alone, 8,000 infants received the B. C. G. at birth. It is further stated that the infant mortality in France has been reduced to one-sixth of the ordinary expected mortality. In England, America, and Germany there has been some fear expressed that this avirulent B. C. G., vaccine might regain its virulent properties after its introduction into the human body, but this has not been found to be the case. How long the immunity from the B. C. G. lasts there has not yet been time to determine, but so far Calmette thinks there is reason to believe that the child is protected against reinfection for over four years. I see no reason why B. C. G. should not be used with the older children and with adults, especially with adults who are exposed to tuberculous infection such as in married life when husband or wife is suffering from open tuberculosis.

How many adults have already received in previous years an immunizing protective infection we do not know. It is the general belief among the most experienced investigators of the subject, however, that the majority have earlier in life received such an infection. It would be easy, however, to determine by the von Pirquet or intradermal test whether any special individual was sensitive to tuberculin (anaphylactic), and if not, then the B. C. G.

could be given if there was to be constant and prolonged exposure to open tuberculosis.

I may remark here that, from my own observation and experience, there is very little danger of contracting active tuberculosis in adult life, if the patient is careful about the disposal of the sputum, and the attendant or one associated with the patient maintains good health.

TECHNIQUE OF ADMINISTERING B. C. G.

The technique of administering the B. C. G. vaccine to an infant is simple, and as given by Calmette, is as follows: (*American Review of Tuberculosis*, June, 1929, Vol. 19, No. 6, p. 567.)

"One-half hour before feeding, the infant is given by mouth a dose of one centigram of fresh culture of B. C. G. in a sterile spoon containing mother's or warm milk. The dose of the vaccine is repeated twice in forty-eight hour intervals. The vaccine can also be given subcutaneously, but this method would seem to be more applicable to older children. The vaccinated child should, so far as possible, be separated from its tuberculous mother (if that is the source of infection) for a period of four weeks, the necessary period to obtain immunity. When this separation is not possible, as may happen, it is necessary to take the greatest precaution to prevent a massive infection during the first month. No general reaction, such as rise of temperature, etc., occurs as a result of the vaccination. 'That the child does not show any allergic reaction (positive von Pirquet)', says Calmette, 'does not indicate that immunity has not been established.' Calmette further states that the Pasteur Institute in Paris will give the original strains of B. C. G. to public health authorities when asked for, and that its use should be controlled by them. In this country Dr. W. H. Park of the health department of New York City has been using this vaccine for some time, and could probably supply it."

THE MILK

One other point in the prevention of infantile tuberculosis, and that is the milk. When the infant is to be artificially fed, the milk should be either sterilized or pasteurized. Even if a tuberculous mother nurses her child, it has been found that the tubercle bacillus is not present in her milk.

PRACTICAL SUGGESTIONS FOR THE USE OF B. C. G.

If further evidence of the value of B. C. G. continues to be as favorable as it now appears to be, why should not public health authorities obtain a supply of B. C. G. which can apparently readily be done, and supply it to physicians and obstetricians, as they now supply antityphoid vaccine and toxin-antitoxin, and circularize them as to the value and use of B. C. G.

As I have shown, the tuberculosis mortality in infants is high and any means which will reduce it, as the B. C. G. vaccine seems to do, is worthy of serious consideration. Moreover, this vaccine, as it has been indicated above, can be used with older children and adults exposed to tuberculosis by intimate association with those suffering with the disease.

THE HIGHEST PEAK IN THE MORTALITY CURVE FROM TUBERCULOSIS

After five years of age the tuberculosis mortality takes a sudden drop. In the registration area of the United States, in 1926, it was 11.9 per 100,000 for the age period of five to nine, and in the age period of ten to fourteen years, 17.6, after 14 years the mortality curve rapidly rises, and from fifteen to nineteen years it is 78.7 per 100,000, and from twenty-five to thirty-four years of age 120.4. At this age period the mortality is higher than that of all the other capital diseases together. After thirty years of age, the curve slowly declines.

THE CAUSE

What is the cause of this high mortality at this age period? Is it an increased susceptibility, a greater opportunity for infection, or a changed mode of life? I believe it is the latter. In early life, young men and women are just entering the strenuous activities of life and selecting careers. Many, perhaps the majority, are entering into industrial or commercial life under exacting conditions. Competition has to be faced. The routine of eight hours a day begins, often under unhygienic indoor conditions. The taking of sufficient and nourishing food is often neglected. Above all, the importance of recuperative rest is not appreciated. After working hours, the young man or woman naturally seeks some diversion and so the evening is devoted to amusement of some kind or other which, in itself, may be a strain on both mind and body. Such may be a night school, moving pictures, a dance, or some other social function. Sufficient sleep is apt to be neglected. As time goes on the young person begins to feel the tire of it all. *Chronic fatigue* insidiously and gradually creeps on. One never feels entirely rested. One gets up in the morning tired, he goes to work tired, and returns home still more tired. So it goes on until a real breakdown occurs which so often takes the form of tuberculosis. I believe there is no more predisposing cause for the development of tuberculosis than *Chronic Fatigue*.

In an analysis of the histories of 1,499 patients having pulmonary tuberculosis, to determine the order of appearance and the relative importance of the various symptoms associated with tuberculosis, the second most frequent symptom which caused the patient to consult a physician was "too easily tired". Cough was the first most frequent symptom. Of the whole number, 1,245 first consulted the physician for

"too easily tired". (Linsly R. Williams, and Alice M. Hill, *Journal of the American Medical Association,* August 24, 1929, p. 579.)

ILLUSTRATION

A young woman, fresh from school and a comparatively free life, takes a position in the city in some office, shop, or factory. She lives in the suburbs or at a distance from her place of work, and either commutes or walks. In consequence, she is obliged to rise early in the morning. She takes a hasty breakfast which she may have to prepare herself. At the noon hour, either from economic or other reasons, she takes a hasty, insufficient lunch. When she returns home, she may have some household work to perform. In the evening, she seeks some diversion—the movies, a dance, or some social function, and finally gets to bed at a late hour. As time goes on she notices that she is losing flesh and feels tired most of the time. *Chronic fatigue* begins. She has a slight breathlessness on exertion; a slight cough—almost unnoticed—appears, and her appetite fails; the earmarks of a beginning active tuberculosis. Such a sequence of events, leading up to the final tragic dénouement, occurs with variation again and again, and it is avoidable. Again, this age period of high mortality from tuberculosis is the period of childbearing, and frequent pregnancies added to the routine duties of the house, make further demands on one's vitality.

DANGERS OF INFECTION

One might adduce another possible cause for this high mortality from tuberculosis at this age period, and that is the greater opportunity of contracting the disease as one, so to speak, enters the great community of people. This, I do not believe, is a factor of very great importance. In the first place, with the adults, the disease is not communicated by a transitory or slight exposure, but only, if at all, by the constant living with one who is suffering from active tuberculosis, has tubercle bacilli in the sputum, and is careless about its disposal.

Physicians and attendants in a sanatorium rarely, if ever, contract the disease.

In the second place, a large number of adults have already been infected, as the von Pirquet test shows, at an earlier age. Many authorities affirm that we all have received this early infection, and that when one does develop active tuberculosis, it is the reactivation of the old infection through the loss of one's normal resistance. Even when a husband or wife is suffering from the disease, authorities differ as to any greater frequency of contracting tuberculosis by the well mate, provided he or she keeps in good condition.

THE REMEDY

If, then, I am correct in believing that this high mortality from tuberculosis at this age period is due to a too strenuous and faulty mode of life, "doing those things which we ought not to do, and leaving undone those things which we ought to do", the remedy is plain—to modify or change one's mode of life, in so far as is possible under the present civilization and competitive struggle for existence. Above all things, the first fundamental factor in the remedy is *REST.* Rest is essentially the main treatment today for pulmonary tuberculosis. If it is so important for the cure of this disease, is it not equally important in its prevention? *Chronic fatigue* must be avoided, and that can be done only by sufficient *rest.*

The conventional working day is eight hours, and there remain sixteen hours for sleeping, eating, going to and from one's work, and time enough besides for sufficient rest, if one would only take it.

On returning home from work, a half hour's rest in the recumbent position before the evening meal is a very valuable relief from the strain of the day's work, especially for young women workers.

Again, a sufficient amount of nourishing food should be taken at regular intervals. Everybody knows, or can easily learn from all that has been said about it, what a properly balanced diet should be.

After the wear and tear of the day's work, the evening, as a rule, should be spent quietly. For example: reading some non-exciting literature in prose or poetry, listening to good music, and so on, will prepare one best for a restful night's sleep of eight or nine hours. "And the cares which infest the day shall fold their tents like the Arabs, and quietly steal away."

The sleeping room should be well ventilated with the windows open.

Of course an occasional evening out, for some diversion, is obviously a natural desire, but this should be of such a nature as not only to divert one pleasantly, but to do so without undue mental or physical over-excitement. I doubt if the movies or the jargon of the radio are conducive to a restful state of mind.

All this emphasis upon *rest,* both of mind and body, may seem a "council of perfection", but the mortality from pulmonary tuberculosis in young industrial life is the highest by far of any age period, and from my observation and experience, *chronic fatigue* is one of the important causes, and the remedy is *rest!*

Some physical exercise is obviously necessary, and, if possible, should be taken out-of-doors. This can readily be done in the longer days of the year after work hours, especially when daylight saving time prevails. One precaution, however, is necessary, namely; when one is thoroughly tired out, whether in mind or body, *rest,* not exercise is indicated. The following observations I made several years ago will illustrate this. (*Journal of the Medical Sciences,* Feb., 1912.) I took the blood pressure of thirty-two young

women in a physical training school, and of ninety-four men taking exercise in a gymnasium. The blood pressure was taken just before the exercise, immediately after exercise, and thirdly, from five to twenty minutes thereafter. As a rule, the blood pressure immediately after exercise was higher than when taken first before exercise, but in five to twenty minutes thereafter, it subsided to what it had been at the first measurement before exercise. On the other hand, if one was tired out, whether mentally or physically, before taking exercise, then the blood pressure after exercise did *not rise* or was lower than at first. For example: an attorney, thirty years old, had a systolic blood pressure of 125 just before "class work" in the gymnasium. Just after his exercise the blood pressure was 112. He gave the history of overwork, both mental and physical. To my interpretation, this indicated that he needed at the time *rest*, not exercise.

The present mode of dress of young women has been adduced as one of the causes of the proportionate increase of tuberculosis in them by some authorities, notably Knopf of New York. I do not believe, however, that this is a factor of any great importance in comparison with the exciting causes mentioned above.

One other suggestion I would make for avoiding *chronic fatigue* and its possible consequences in the development of tuberculosis, and that is a longer vacation, especially for young women engaged in continuous work in industrial or commercial life. The conventional two weeks' vacation is not enough; it should be a month spent quietly by the seaside or in the country, or it might be divided into two portions, one in the summer and the other in the winter. Home making would seem to be the intended career of women, but modern conditions of civilization and necessity have forced them into industrial occupations for which many are unfitted. Moreover, a month's vacation for such workers would, I believe, enable them to do better work and more than compensate for the extra two weeks.

Professor Fisher (quoted by Dr. Patterson) says, when discussing the effect of the long working day on the health of workmen, "A typical succession of events is, first fatigue, then colds, then tuberculosis, then death."

When cases of chronic fatigue and breakdown occur in young women workers, a preventorium such as now exists for children, would seem to be a most excellent provision. The writer visited just such a one, at Ste. Agathe in the Laurentian Mountains of Canada, founded by the late Dr. Richet.

TO SUM UP

Sufficient *rest;* nourishing and sufficient food; some outdoor life and sunshine; good ventilation in working places and at home; these are the fundamentals for preventing tuberculosis at this critical age period.

It goes without saying that at the same time, the habits of personal hygiene should be observed.

PNEUMOKONIOSIS OR SILICOSIS

The granite cutting industry in New England is extensive, and large numbers of men are employed in it. One of the most prevalent diseases arising from the inhalation of silica (granite) dust is pneumokoniosis which so often finally results in chronic pulmonary tuberculosis (Stone Cutter's Phthisis). McFarland, of New Jersey (*Industrial Hygiene*, August, 1927, IX, 315), reports the death rate from tuberculosis among granite cutters as 1,095.5 per 100,000 which is about eleven and a half times the average rate. In 1922 Hoffman found that among the granite cutters in Vermont, the mortality rate from pulmonary tuberculosis was 1064.5 per 100,000 as compared with 108.3 for the general population for the state of Vermont. It seems an irony of fate that while the workman is fashioning the stately mausoleum in granite for the great or rich, he is, at the same time, digging his own grave.

It has been found that if the granite worker leaves his occupation, after a short time, the silica deposits in his lungs may disappear, but at the end of seven years, according to Pitchford (*Industrial Hygiene* for 1927, IX, 109), a little more than half of the cases of simple silicosis have become tuberculous. Much of this excessive mortality in stone cutting, I believe, can be prevented by periodic examinations of the workmen; short working hours; the avoidance of dry drilling, and use of masks and compressed air apparatus for carrying away the dust; and the removal from the industry as soon as any appreciable amount of deposit is found in the lungs; and by the use of various apparatus for carrying off the dust, some of which have already been devised and are in use.

TYPHOID FEVER

Although the mortality from typhoid and paratyphoid fever is the lowest of any disease with one or two possible exceptions, still there are always a few cases—for example in 1928 there were ten cases in New Hampshire or 2.2 per 100,000, and in Massachusetts in 1927 there were 42 cases or 1.0 per 100,000. Improved sanitation, careful milk and water inspection, and the antityphoid prophylactic vaccine have been the cause of this almost complete disappearance of typhoid fever, which, not so long ago, produced a large yearly mortality—for example, in 1900 the death rate from the disease was 36 per 100,000 in the registration area of the United States. Still there is always the possibility that any of us may be of the unfortunate few who contract the disease, and yet it is so easy to avoid that possibility by

means of the prophylactic use of antityphoid vaccine, the protection afforded by which the great war has so thoroughly established. It is now the common custom for one contemplating a visit to Europe to take the three antityphoid vaccine injections on account of the difficulty of obtaining pure drinking water abroad, and the hazard of drinking water of unknown source. Does not this hazard, at least to some extent, exist while traveling in this country, especially on automobile tours so common at this time? Would it not, therefore, be an equally reasonable precaution to take the antityphoid prophylactic vaccination, the material for which can so easily be obtained from any state laboratory?

To illustrate: a case came under the observation of the writer of a gentleman who took an automobile vacation tour, and who contracted typhoid fever from some unknown source on the route, and returned home and died. He happened to be one of the unfortunate few, and the tragedy might have been avoided if he had taken the precaution of antityphoid vaccine protection.

DIPHTHERIA

It is common knowledge that the mortality from diphtheria has been vastly reduced since the introduction of the antitoxin treatment. In New Hampshire in 1928, there were but 26 deaths or 4.4 per 100,000, and in Massachusetts, in 1927, 275 deaths or 6.5 per 100,000.

The morbidity is unknown but must be much greater. It has also been stated on reliable authority that from 60 to 70 per cent. of deaths occur in children under five years of age. It has been absolutely demonstrated that toxin-antitoxin will give immunity to this disease, and in many communities this means of prevention is already being employed, but apparently more extensively in the school than the preschool age. In most communities the public health authorities require evidence of vaccination against smallpox before admitting children to the public schools. Is there not equal reason why they should not be vaccinated against diphtheria "which still persists as one of the serious infections"?

SCARLET FEVER

The mortality from scarlet fever in New Hampshire in 1928 was 20 or 4.4 per 100,000, and in Massachusetts, for 1927, 145 or 3.4 per 100,000. The morbidity, of course, is much greater.

Although the scarlet fever streptococcus vaccine may not be advisable for routine use, yet when there is a case of the disease in the household, or an epidemic in the community, it would seem to be a wise precaution to employ it, particularly when one considers the possible serious complications of this disease such as acute nephritis and mastoiditis. The so-called Dick test to determine one's susceptibility to the disease is a skin test applied in the same way as the Schick test for diphtheria. Although it may not always be reliable, yet when there is direct contact or exposure to the disease, this test should be applied and if positive, the scarlet fever streptococcus vaccine should be employed with children under 12 years of age. There should be no difficulty in obtaining the material for the test and vaccine from state laboratories.

HEART DISEASE

As will be seen in the chart of the distribution of the six principal diseases by age groups, the curve for the diseases of the heart begins to rise quite abruptly at forty years of age, and reaches its peak from sixty-four to seventy years of age—a peak higher than any of the other five capital diseases. At this age period, moreover, while the expectancy of life from birth has increased, as has been stated above, about 43 per cent. in the last hundred years, the expectancy of life of one who has reached fifty years of age was 21.2 years longer a hundred years ago, while it is but 21.5 years longer at the present time. In fact it has been asserted on good authority that the expectancy of life after middle life is actually decreasing. To heart disease, which leads all the others in mortality (96,682 deaths in the registration area of the United States in 1926) must be attributed the largest share of this practically stationary or diminishing expectancy of life after fifty years of age for the last century. In other words, a man has arrived at the degenerative stage of life too quickly; he is wearing out his heart too soon.

ILLUSTRATIONS

(1) A man of middle age or a little over, walking in the street, suddenly falls dead from "heart failure". (2) A clergyman of about the same age of sedentary habits walking against a strong wind develops acute oedema of the lungs and dies, due again to weakness of the heart. (3) A literary man of less than middle age accustomed to strenuous mental work, without physical exercise, while swimming during his summer vacation suddenly succumbs to heart insufficiency. (4) A retired business man suddenly drops dead on the golf links—and so the list might be indefinitely extended.

Why should so many persons die of heart insufficiency before they have lived out the allotted biblical span of life of three score years and ten? Obviously the cause is the mode of life the individual is pursuing. As Prof. Forsyth of Dartmouth College says in a recent article in *Science*, "The vast majority (of adults) seem very indifferent and many give apparently no thought whatever to habits which they clearly know are bad and which they know they could easily discard." It is the misuse and neglect of the

engine (the heart) of the human machine, with little regard to its limitations and care. One can use up his automobile in a year, or with proper care it will last many years.

The modern man of affairs works his heart too hard or from another point of view, he does not work it enough. The automobile will degenerate if kept in the garage and never used. The heart needs both rest and exercise to maintain its integrity. In what I am saying I am, of course, leaving out of account those defects of the heart caused by infectious diseases, and by rheumatism and syphilis. The condition of one's heart at sixty and later years of age will depend largely on how he has treated it in earlier years.

PREVENTIVE TREATMENT OF HEART DISEASE

In the first place one's general mode of life from the first must be hygienically correct. As I have said, the heart needs both rest and exercise, and proper nervous control. If one's occupation is largely physical more rest is needed, comparatively, than for one whose occupation is sedentary. At the age period from 60 to 70, when the mortality peak is so high, a certain amount of daily rest is desirable, preferably taken in the recumbent position so as to lessen the "load".

Equally desirable is exercise adapted to the condition and age of the individual, as well as to his tastes.

I recently saw a college professor of 80 years or more, who takes his daily exercises now on the river, rowing.

It is to be remembered that the heart is a muscle, and like other muscles in the body, becomes flabby without some exercise in addition to its routine work. Likewise the nervous system through which the heart movements are regulated, must also have rest and freedom from over-excitement and stimulation. The diet should be moderate, with little meat or purely vegetarian. If one has been used to it, a moderate amount of alcohol in the form of wine or beer is allowable, or indeed may be beneficial, taken at meal times. I recall the remark I heard Osler once make in his clinic to a patient, that after 60 years of age he could take a little alcohol with benefit, but none before.

Of course the time will come as age advances, when degenerative changes in the heart will take place as in other organs, dependent to a certain extent upon heredity, as well as upon age. One of the not infrequent conditions of the degenerative changes is AURICULAR FIBRILLATION often unobserved by the individual until well established. If untreated this condition may go on from bad to worse to a fatal termination, but with early discovery and proper treatment, it may not perceptibly shorten life.

TREATMENT OF AURICULAR FIBRILLATION

First much rest must be employed, perhaps complete rest in bed, according to the condition. At the same time, digitalis of a standardized and reliable make is given in sufficient quantity to reduce the pulse to 80 or below, and thereafter the digitalis should be continued indefinitely to maintain the pulse at this rate. It may be 1½ grains of the powdered leaf equivalent to 15 minims of the tincture, once or twice a day, or even less. If the pulse remains at or below 80 beats, the digitalis may, at intervals, be omitted altogether. Unless there is some contraindication, the usual diet, if moderate, may be taken. As conditions improve a moderate amount of exercise can be taken on the level, but one had best avoid stairs or up grades unless taken very slowly.

Rest, digitalis wisely given, and a quiet routine of life, will do wonders for auricular fibrillation unless too far advanced. An excellent means of discovering heart defects at an early stage would seem to be periodic health examinations which has been preached for the last two or three years. From my observation, however, this invaluable health protective measure has not met with such response from the public as it deserved or was hoped. In the first place it has not been sufficiently or adequately advertised to the public. This is an era of advertising, and the public depends on much reiterated presentation of any measure offered to it, to become consciously aware of it. In the second place the general practitioner who would naturally do most of such examinations, was given too much detail to determine. He was presented with a long formula equivalent to that for the examination of an applicant for the military service, and he did not feel competent to make such a complicated examination. Furthermore, it is doubtful if he was enthusiastic about undertaking such an elaborate examination requiring an hour or more, for the fee that the average individual was willing or able to pay, since the average man is not conscious of any physical defect. I would suggest that the physician should confine himself simply to an examination of the heart, lungs, and kidneys which he can do with a fair degree of accuracy, and in a comparatively short time, and then if he is doubtful about the result of his examination, or if a more detailed one is desired, the individual can be referred to a specialist, or to a health clinic—so-called. For example, such a health clinic is now conducted at the Boston Dispensary, where a group of specialists make the examination and where they have the aid of laboratory facilities and the x-ray. A complete examination of this sort is done at the small fee of $7.50 with a small additional charge for laboratory and x-ray examinations when these are necessary. It is a pity that this most valuable means of discovering disease in its incipiency should not have a greater vogue than it now seems to have.

PNEUMONIA

It will be seen from the chart of the six capital diseases that pneumonia has two high peaks,

at different age periods, the one in infancy and the other in the later years of life. In the registration area of the United States for the year 1926 there were 80.5 deaths per one hundred thousand inhabitants from pneumonia, of all ages, and all forms. Under one year of age there were 1,066.4, and under five years of age 320.7 per one hundred thousand. The mortality continues low up to fifty-five years of age, when between the ages of fifty-five and sixty-four it is 145, and between the ages of sixty-five and seventy-four it is 234, and from seventy-five years and over, it is 441 per one hundred thousand. In New Hampshire in 1928 the mortality from all forms of pneumonia, at all ages, was 424, or 93 per one hundred thousand.

Thus, we see that there are two periods in the life span when pneumonia has its greatest mortality, one in infancy and the other in the later years of life. The greatest mortality, however, as we have seen, is in infancy, and the usual form of pneumonia at that age is broncho-pneumonia. These two extremes in the mortality of pneumonia might be called a possibility as with the infant, and an accomplished fact in old age when one's life work has been done. Incidentally, compare this with the highest mortality age period of pulmonary tuberculosis which is at the beginning and summit of one's best years of economic life. The greatest mortality from pneumonia occurs in the colder months of the year; with infants it is in February and March. This is probably due more to the results of indoor life in impure and overheated dry air than to outdoor exposure.

The fundamental cause of this high mortality at both extremes of life is essentially the same, namely, the lack of resistance. In the case of the infant, resistance has not been developed, and in the case of the old man it has been lost, or greatly reduced. The prophylactic measures obviously then, are to develop resistance in the one, and to maintain it in the other.

Prophylactic pneumococcic vaccine was tried in some of the Army Camps during the great war, and apparently with some degree of success, but so far as I am aware it has never been employed subsequently with any large numbers. A division of troops, 12,519 in number, were immunized with a vaccine containing pneumococci of types 1, 2 and 3 of pneumonia. No case of any of these three types developed while a corresponding body of men unvaccinated developed numerous cases of types 1, 2, and 3 pneumonia. (*Journal of Experimental Medicine*, July 1, 1918.) The types of pneumonia are so varied that it seems doubtful if any one vaccine would give protection.

GENERAL PROPHYLACTIC MEASURES IN INFANTS

Much, I believe, can be done to prevent this enormous mortality from broncho-pneumonia in infants. It depends largely, as I have said, on the development of the child's resistance. It may be called a hardening process. First, the child needs fresh air and sunshine; with proper protection he can be much out-of-doors in all but the coldest weather of the year, and even then in the sunshine. When indoors, the room where the child is should be well ventilated with a temperature not above 70 or 75 degrees, and a relative humidity not below 30%. Secondly, the food should be properly prepared as to its nutritive qualities and in sufficient amount. There are now in many towns baby clinics, as well as much literature whereby knowledge can be obtained as to the proper care and preparation of infants' food. It goes without saying that the most perfect food for the baby is its mother's milk, but the modern mother seems to lack the ability or is disinclined to nurse her child. Third, *rest*. There is no more important means of developing increased resistance in the child than rest. The baby naturally sleeps a good part of the twenty-four hours, and care should be taken to see that it obtains its full quota of sleep and rest. The growing young of all animals sleep much. Rest for the nervous system is equally important. The exhibition of the baby by doting parents is a tax upon its nervous system and a pernicious practice. The clothing of the child should be just enough to keep it warm and comfortable. The child should be protected from much fondling from strangers, and should not be promiscuously kissed. If these fundamental principles in the care of the child were rigorously observed, I believe there would be less mortality from pneumonia at this age period.

PREVENTION OF PNEUMONIA IN ADULT AND LATER LIFE

As with other infectious diseases, a temporary, or chronic fatigue is, I believe, a frequent predisposing cause of pneumococcus infection, particularly with adults after early middle life. For example, a strong man of about forty-five years of age, a salesman, had a particularly strenuous life for a week previous to his illness, traveling in cold sleeping cars at night, and working hard during the day. At the end of the week he developed pneumonia from which he died. He had temporarily lost his resistance. Second, a lawyer in poor condition and having a cold, thought he could work it off by taking strenuous exercise. He too, developed pneumonia which proved fatal. What he needed was rest and not exercise. In both cases there was a temporary loss of resistance, the result of over-fatigue.

A man at fifty years of age who is "healthy" and has no obvious disease, may expect to live twenty years longer. The only way to fulfill this expectation and to avoid the frequent and dangerous disease of pneumonia, is to keep healthy and avoid over-fatigue. If a period of temporary over-fatigue occurs, which is sometimes unavoidable, there should follow a

period of rest. Sometimes what appears to be a common cold in the beginning culminates in pneumonia, and if one should follow the advice often given, but less frequently followed, of going to bed at the beginning of the cold and remaining there several days, this possibility would be less likely to follow.

PNEUMONIA IN THE LATER YEARS OF LIFE

The curve of mortality in pneumonia is high in the later years of life, from sixty to eighty years of age. This is the degenerative period when resistance to disease is failing. Much depends, however, upon how one has treated his organs in previous years, and especially upon the condition of one's heart.

PROPHYLACTIC MEASURES

Avoidance of over-fatigue, rest, pure air, exercise suitable to one's age, and a proper diet are the cardinal principles to be observed in the avoidance of pneumonia in the aged. As age advances, one needs more rest both of mind and body. One must reduce his hours of work and make them less exacting. One needs some exercise out-of-doors when the weather permits, but well within the limits of one's strength. The diet should be nourishing but frugal. Overeating is sometimes a fault of the aged. A little alcohol at bedtime or at dinner, I believe, is of value, at least in some cases, in stimulating the waning forces of digestion. It is an increasing custom for the elderly person to seek a warm climate in the colder months of the year, in Florida or southern California. Undoubtedly such a change has its advantages, for it gives a greater opportunity for life in the open. With such a mode of life, however, and with such precautions as I have indicated above, I believe the elderly person would do about as well in a climate to which he has always been accustomed and with the comforts and precautions of home surroundings. Moreover the change from one climate to another is abrupt and one may meet with colder weather than one anticipates in returning north.

THE COMMON COLD

In a communication issued by the United States Public Health Service, by Assistant Surgeon General Rucker, his introduction is the following sentence, "The most prevalent illness in the United States is the common cold, a disease group included under one name and considered of such minor importance that vital statistics do not record the enormous numbers of persons who annually are subjected to suffering, inconvenience, and economic loss thereby. Remarkable as it may seem the widespread familiarity with this condition has bred contempt which hides its seriousness and yet when the sum total of the ravages committed by common colds is made, it becomes evident that instead of being a group of trivial affections, common colds must be classed as serious diseases."

A cold may lead to pneumonia, sinus disease, aural disease, or bronchitis, and one cold renders one more susceptible to another. If such is the case as stated above, it is worth while to consider seriously what can be done to prevent a cold.

PREVENTION

In the first place, colds are infectious, and spread from one person to another, therefore every effort should be made to avoid this infection, first, by keeping away from one who has a cold and coughs or sneezes, and at the same time admonish him who coughs or sneezes to hold something before his mouth when he coughs. Again, one should not use toilet articles which have been used by others, such as towels, drinking cups, etc.

A cold may well be called a disease of indoor life, for colds are by far the most frequent in the colder months of the year, when one lives largely an indoor life, hence it is evident that to regulate properly one's indoor life is one of the important means of preventing colds. The atmosphere indoors must be pure, sufficiently moist, and of proper temperature. The living room or the office should be "aired out" by opening the windows one or more times a day. The temperature should be about 70 F., and quite as important as a thermometer, is a hygrometer to determine the relative humidity which should be not below 30% at the minimum. In the majority of artificially heated rooms it is below this. As someone has said, the air is as dry as the driest desert in the world. Some method therefore should be employed to keep the air moist in the living room and in the bedroom, and this can readily be done by means of receptacles containing water placed over the radiator or on the stove. The best means of doing this is an unglazed vessel like a flower pot, from which the water will evaporate not only from the surface, but through the sides.

Besides environmental hygiene, personal hygiene is important. One needs more rest in the stimulating winter weather and generally one does not take as much in sleep. One needs proper and sufficient food, exercise in the open air, and sunshine. In brief, to keep the body resistance to a high degree of efficiency is the best preventive of colds.

As to the use of vaccines for the prevention of colds, the evidence is conflicting. Speaking from personal observation and experience as to cold vaccines, many of which are now in the market, it may or may not be successful. A trial in individual cases only can determine the result. A more certain method of prevention is personal and environmental hygiene and the avoidance of infection. I believe that a cold is a preventable disease and it is serious enough to employ every means to prevent it.

CONCLUSION

In the foregoing, I have attempted to indicate some of the measures for the prevention of disease and have made some suggestions as to their greater utilization.

The prevention of disease is one of the great movements of the present day. Much already has been accomplished in banishing, or greatly reducing many diseases, as has been seen. At the Twenty-fifth Annual Meeting of the National Tuberculosis Association at Atlantic City in May, 1929, Opie, in his presidential address, concluded with these words, "Health is purchasable, if we knew what to buy." (*American Review of Tuberculosis*, August, 1929.) I would add that we already have a great deal of knowledge of what to buy, if we would only utilize it. The problem is to induce both the medical profession and the public to realize its value. It largely depends upon the medical practitioner to do his share in this greater movement in preventive medicine. Preventive medicine should be taught in the medical schools, in the clinics, and by the bedside. The physician should ask himself and his students, "Could this disease have been prevented, and how?" Furthermore, general information should be disseminated among the general public as to the importance of prevention. There is a rapidly increasing amount of publicity of an authoritative kind, with regard to the prevention of disease, in magazines, the daily papers, and the radio. Still more is needed on this vitally important phase of health protection. The physician by his contacts in his community can probably do more than any other instrumentality. He must, however, thoroughly realize the present state of preventive medicine and familiarize himself with the measures already at hand for preventing disease. "Principiis obsta" should be his watchword.

MISCELLANY

CARROLL COUNTY MEDICAL SOCIETY

A meeting of the Carroll County Medical Society was held at the Center Ossipee Inn on the evening of November 13. The program follows:

A County Pre-Natal Conference—F. E. Clow.

Some Impressions of General Practice—Charles Smith.

Medical Education as it Affects Carroll County—E. H. Egbert.

A full discussion followed, particularly with regard to the first topic. It was voted that the proposition, as outlined by Dr. Clow, for a monthly prenatal conference at the County Hospital, with the coöperation and support of the county commissioners be accepted and heartily supported. One physician will devote a part of an afternoon to the examination of prospective mothers and, with the assistance of public health nurses, seek to guide them during a critical period. The Board of County Commissioners has, with unusual foresightedness and generosity agreed to assume the financial burden, and provide rooms for the conferences, in the belief that it is only with adequate ante-natal care that the maternal and infant mortality can be lessened. Carroll County is to be congratulated on having a board of commissioners with vision to realize the possibilities of such a measure, the first of its kind in the state.

The following officers were elected:

President: Edward H. Egbert, Wolfeboro.

Vice President: Edwin C. Remick, Tamworth.

Secretary-Treasurer: Chas. Smith, Conway.

Censor: Fred E. Clow, Wolfeboro.

Delegates to New Hampshire Medical Society: Edwin C. Remick, Tamworth; Charles Smith, Conway.

ROCKINGHAM MEDICAL SOCIETY

At the annual meeting of the Rockingham Medical Society, held in Derry, New Hampshire, in October, the following officers were elected for the ensuing year:

President: Dr. Walter R. Sanders, Derry Village, N. H.

Vice-President: Dr. T. C. Tappan, Derry, N. H.

Secretary: Dr. John G. W. Knowlton, Exeter, N. H.

Treasurer: Dr. W. P. Clare, Portsmouth, N. H.

Delegate: Dr. Eugene P. Eastman, Portsmouth, N. H.

Alternate: Dr. L. R. Hazzard, Portsmouth, N. H.

County Committee Member on Medical Defense: Dr. T. W. Luce, Portsmouth, N. H.

Committee on Public Policy and Legislation: Dr. T. W. Luce, Portsmouth, N. H.; Dr. C. W. Hanniford, Portsmouth, N. H.; Dr. Abraham Mitchell, Epping, N. H.

NEW MEMBERS

Dr. Donald W. Leonard, Exeter, graduate of Harvard Medical School, 1929.

Dr. George L. Fox, Exeter, graduate of McGill University Medical School, 1929.

Dr. Donald C. Moriarty, Groveton, graduate of University of Vermont Medical School, 1929.

Dr. Raymond J. Turley, Berlin, graduate of University of Vermont Medical School, 1929.

Dr. Fred A. Sprague, Concord, has rejoined the Society after an absence from the state since 1917 when he was commissioned captain in M. R. C.

DEATHS

Dr. Fred Ellsworth Bryar of Concord, formerly of Centre Sandwich but for the past thirteen years an assistant member of the staff of the New Hampshire State Hospital, died at Brighton, Mass., October 25, after a brief illness.

Dr. B. Frank Horne of Conway died at his home October 25. For some years he was Secretary of Carroll County Medical Society and Medical Referee, holding both offices at the time of his death.

Dr. James Joseph Buckley of Dover died at his home November 4 after a long illness. He had been the efficient Secretary of the Strafford County Medical Society.

VERMONT STATE MEDICAL SOCIETY

HOUSE OF DELEGATES

THE fixed session of the House of Delegates was called to order by the President, Dr. L. A. Russlow, at 5 P. M., Thursday, October 2, 1930, in Lecture Hall "A" of the Medical College Building, Burlington, Vt.

The secretary called the roll, and by vote of the House vacancies therein were filled, making the roll of the House as follows:

Addison County:—P. L. Dorey.
Bennington County:—Charles S. Buchanan, John D. Lane.
Chittenden County:—C. A. Ravey, Sam Sparhawk.
Franklin County:—Edwin Hyatt, G. A. Abell.
Lamoille County:—W. M. Johnstone.
Northeastern:—F. E. Farmer.
Rutland County:—E. J. Rogers, N. B. Lanou, H. L. Frost, B. F. Cook, J. E. O'Brien.
Washington County:—F. C. Angell, M. F. McGuire, L. B. Allen, L. A. Russlow, J. A. Wark.
Windham County:—J. A. Stevenson, —. —. Bowen.
Windsor County:—A. M. Cram, B. A. Chapman.

The report of the Secretary* was called for, and Dr. W. G. Ricker gave the substance of the proceedings of the 1929 meeting of the House of Delegates.

The president called for reports of committees, and on motion of Dr. Rogers, seconded, it was voted to accept the various reports as printed, without being read in the House of Delegates.

Under "Unfinished Business," the Secretary stated he knew of no unfinished business.

Under "New Business" the place of the 1931 meeting was taken up and Dr. J. A. Stevenson moved that the Society hold its 1931 meeting in Rutland, which motion was seconded and carried.

Dr. W. G. Ricker, not being a member of the House, was accorded the privilege of the floor, and brought up the subject of proposed changes in the Workmen's Compensation Law, suggesting that members of the Legislative Committee be most carefully selected, who would make it their business to get in touch with secretaries of county societies, and ascertain what the county societies wished; such legislative committee to meet shortly before the convening of the next legislature.

Dr. McGuire outlined what he understood to be the wishes of the Commissioner of Industries in regard to adequate pay for medical services.

Dr. McGuire moved that a committee of three be appointed by the Chair to discuss the subject and enlist any others they wished, in the discussion of the matter.

Motion seconded.

The subject was further discussed by Doctor Lane, Doctor Bowen, and Dr. Wark.

Dr. Ricker stated that if such a committee were appointed and wished to hold a meeting, that he would have notices sent to everyone in the state who might be interested, that the committee was meeting on such a date, at such a place.

At the request of Dr. Hyatt the motion of Dr. McGuire was read.

Dr. Stevenson offered an amendment to Dr. McGuire's motion to the effect that this committee meet in Burlington around the first of December, and everyone interested be sent notices, and those wishing to be heard should meet with the committee.

Amendment accepted by Dr. McGuire.

The subject was further discussed by Dr. Buchanan and Dr. Cram.

Dr. Rogers expressed the opinion that Dr. McGuire's motion was out of order inasmuch as the society has its legislative committee, which could call in men from each county.

The motion of Dr. McGuire, as amended, was put, and voted down.

Dr. Johnstone suggested that the matter of a special committee, and the subject of compensation be left with the secretary and the nominating committee, to bring in suggestions at the adjourned meeting of the House of Delegates, on Friday morning.

Dr. Cram, Dr. Lane and Dr. Wark further discussed the subject.

Mr. H. T. Corson, Executive Manager of the National Food Bureau, was given the privilege of the floor for ten minutes, and outlined work which the Bureau was doing in other states, in bringing before the public the beneficial uses of wheat flour, and offered the following resolution:

Whereas: Much misinformation is promulgated today concerning the question of diets, thus causing the introduction of food fads, very few of which can take the place of the older staple foods; and whereas,

Any balanced diet should contain animal protein, fruits, vegetables, especially the leafy vegetables, and the better grades of bread prepared from flour which will insure adequate vitamin and mineral salt content, digestible fat such as butter-fat, and sufficient of the digestible carbohydrates to afford readily available energy; and, whereas,

The allegation that white bread, meat or any other staple food, when employed in mixed diet is responsible for certain grave illnesses, is not supported by scientific facts,

Therefore, Be It Resolved That: We desire in the public interest, to place on record that in our opinion:

1. The exaggerated claims for various fad foods are unwarranted by scientific evidence or practical experience; and the advertising and other propaganda furthering their substitution for the older articles of diet should be condemned.

2. The danger of nutritional deficiencies has been grossly exaggerated. No one food is a perfect food; but a diet consisting of dairy products, leafy vegetables, fruits, meats and easily digested starches furnishes an excess of all food factors

*See page 1156.

necessary for proper growth and nutrition and resistance to disease.

3. Any variation from a normal diet should be prescribed only by a properly trained physician after a careful study of the dietary requirements of the individual seeking advice.

Dr. Hyatt moved that the resolution read be received by the House of Delegates and laid upon the table. Motion seconded and carried.

Dr. Angell moved that the legislative committee soon to be elected be urged to give special attention to the Workmen's Compensation Law relative to the pay for medical services.

Motion seconded and carried.

Dr. Allen, of Post Mills, moved that the State Medical Society pay the expenses of the legislative committee. Motion seconded and carried.

The President called for nominations for president of the Society for the ensuing year.

Dr. Johnstone tendered his resignation as a member of the House of Delegates, which resignation, on motion of Dr. Buchanan, seconded and carried, was accepted.

Dr. F. E. Farmer nominated Dr. W. M. Johnstone for president of the Society for the ensuing year. Nomination seconded by Dr. Stevenson, and Dr. Johnstone declared elected President.

The President called for nominations for secretary, and Dr. Stevenson nominated Dr. Wm. G. Ricker. Nomination seconded by Dr. Wark, and Dr. Ricker declared elected Secretary.

Dr. Stevenson moved that a committee of three be appointed by the Chair to bring in nominations for the remaining officers and committees, at the adjourned session of the House, to be held Friday morning, and 3rd inst., at 8:30. Motion seconded and carried.

The Chair appointed as such nominating committee, Dr. Stevenson, Chairman, Dr. Lane and Dr. Farmer.

Moved by Dr. Stevenson that the House of Delegates adjourn until 8:30 Friday morning, the same place.

Motion seconded and carried.

The President declared the House of Delegates adjourned until 8:30 Friday morning, Oct. 3, 1930, same place.

Friday Morning Session

The adjourned meeting of the House of Delegates was called to order by Dr. L. A. Russlow, President of the House, at 8:30, in Lecture Hall "A" of the Medford College Building, Burlington, Vt., Friday morning, October 3, 1930.

Pres. Russlow called for the report of the Nominating Committee.

Dr. Stevenson, Chairman of the Nominating Committee, gave the following report:

President, Vermont State Medical Society: William M. Johnstone of Morrisville.

Vice President: Lyman Allen, of Burlington.

Secretary: William G. Ricker, of St. Johnsbury.

Treasurer: David Marvin, of Essex Junction.

Auditor: E. H. Buttles, of Burlington.

Councillors:
1st District—T. A. McCormick, of St. Albans.
2nd District—John Trotter, of Bennington.
3rd District—F. E. Farmer, of St. Johnsbury.
4th District—John Woodruff, of Barre.

Executive Committee: William G. Ricker, Chairman, St. Johnsbury; S. W. Hammond, Rutland; E. L. Tracey, Brattleboro.

Publications Committee: William G. Ricker, Chairman, St. Johnsbury; C. F. Dalton, Burlington; E. A. Stanley, Waterbury.

Legislative Committee: John D. Lane, Chairman, Bennington; J. A. Wark, Secretary, Barre; A. M. Cram, Bridgewater.

Medical Education: S. W. Hammond, Rutland; A. B. Bisbee, Montpelier; A. C. Black, Brattleboro.

Necrology Committee: L. A. Russlow, Randolph; B. D. Adams, Burlington; H. L. Pierce, Swanton.

Medico-Legal Committee: F. E. Farmer, St. Johnsbury; E. A. Hyatt, St. Albans; J. H. Jenne, Burlington.

Health and Public Instruction Committee: P. E. McSweeney, Burlington; A. L. Patch, Windsor; C. F. Dalton, Burlington; A. C. Walker, Springfield; P. K. French, Burlington.

For officers of the House of Delegates:
President: E. A. Hyatt, of St. Albans.
1st Vice President: E. J. Rogers, of Pittsford.
2nd Vice President: P. L. Dorey, of Middlebury.
Secretary: Naomi Lanou, of Rutland.

Delegate to American Medical Association: William G. Ricker, of St. Johnsbury.

Alternate to American Medical Association: G. H. Beecher, of Burlington.

Anniversary Chairman: George R. Anderson, of Brattleboro.

For Delegates to State Societies:
Maine: Frank E. Farmer, St. Johnsbury.
New Hampshire: A. L. Patch, Windsor.
Massachusetts: John Blodgett, Bellows Falls.
Connecticut: C. S. Buchanan, Bennington.
Rhode Island: Arthur B. Woodman, Springfield.
New York: L. H. Ross, Bennington.

New England Medical Council:
G. F. Dalton, Burlington.
William G. Ricker, St. Johnsbury.
W. M. Johnstone, Morrisville.
J. A. Stevenson, Chester.
E. A. Hyatt, St. Albans.

Moved that the report of the Nominating Committee, as submitted, be accepted. Motion seconded and carried, and the President declared the nominees elected to the respective offices for which they were nominated.

Dr. Buchanan moved that the Society pay the sum of twenty-five dollars to Dr. Ricker's private secretary for additional work which she had performed in connection with the 1930 Medical Society meeting.

Motion seconded, and carried.

Dr. Stevenson moved that the House of Delegates adjourn.

Motion seconded and carried.

Adjournment.

SECRETARY'S REPORT

To the Members of Vermont State Medical Society

"Come over into Macedonia and help us" is the cry underlying some of the County reports which are appended hereto.

It is true that one year ago some funds were available, enabling the State Society and State Board of Health together to send a team of four men through the State and from the reports received these meetings were successful. An attempt was made this year to hold such meetings again, but on account of various factors the plan did not succeed.

The suggestion of the supervision of district visits such as is carried on by Rotary and other organizations is certainly a wise one, but unfortunately the officers of the State Society are all in active practice and it is certainly difficult to find the time to carry out such a scheme.

One thing, however, is certain, no County Society can succeed by being bottle fed by the State Society. Its own officers must call its meetings, and when and if they wish Officers and other members of the State Society to attend, an invitation should be sent, and so far as humanly possible, this invitation will be accepted. The President of each County Society must take his duties seriously and not be satisfied, simply by presiding at called meetings. He should be active with the Secretary to see that meetings are held.

Moreover, the meeting habit should be formed and fostered. Meetings held at hit or miss times do not excite interest, but when held at fairly regular and frequent intervals (say four times per year) acquaintanceship is formed and there develops a spirit of comradeship and coördination within the group.

An ex-President of the Canadian Medical Association has said to me personally, that he considers the social features of a medical meeting of greater importance than the scientific program, and I agree with him. The scientific program is not only important, but necessary, and should be prepared with the greatest of care, for every physician, worthy of the name, is seeking further information. But, in addition to that, coöperation of the closest sort must exist, if our patients are to receive the full benefit from us to which they are entitled.

A careful perusal of the reports from County Societies will indicate the varying success of each and will offer to all members various suggestions.

William G. Ricker, M.D., *Secretary.*

September 24, 1930.

Supplement to Secretary's Report

	Membership, 1929	Membership, 1930	Increase	Decrease
Addison	14	13		1
Bennington	20	21	1	
Chittenden	75	74		1
Franklin	28	28		
Lamoille	9	8		1
Northeastern	43	42		1
Rutland	50	55	5	
Washington	52	52		
Windham	28	26		2
Windsor	22	21		1
	341	340	6	7

REPORTS OF COUNTY MEDICAL SOCIETIES

Addison County

Aside from the list of officers no report was submitted.

Bennington County

Annual meeting and election of Officers for 1931 will be held after state meeting. Regular monthly meetings of Society will begin the last Wednesday of September, 1930 and continue until June, 1931. Microscopy and postmortem work will receive special attention this coming year.

Frank J. Hurley and family recently returned from an extensive vacation tour of the Rocky Mountain region. Doctors Wilson, Ross and Trotter recently returned from vacation trips. Dr. Lane and wife will sail from New York, September 6, for a two weeks' vacation voyage.

J. D. Lane, M.D., *Secretary.*

Chittenden County

Seven regular meetings were held during years 1929 and 1930, and one joint meeting with the Rutland Ry. Surgeons.

Papers:

Post Vaccine Encephalitis—Dr. E. W. Pike.
Symptoms both direct and reflex, of intra-abdominal lesions—Dr. Lyman Allen.
Medicine and aviation—Major Stanley.
The use of Calcium Chloride in Acute Epididymitis—Dr. Seth Martin.
Present day treatment of Tuberculosis, especially in Sanitariums, particularly at Pittsford—Dr. E. J. Rogers.
Pelvic Inflammation in the Female—Dr. O. N. Eastman.
Tumors, their diagnosis and prognosis—Dr. Charles Branch.
Traumatic rupture of the urethra, associated with fracture of the pelvis, by Dr. Vander Veer, Albany, N. Y., read by Dr. John Horner.

Average attendance was 25, an increase of two over preceding year. We have 73 members in the Society, all in good standing. Two new members were elected; one dropped and two died during the year.

Ralph G. Streeter, *Secretary.*

Franklin County

After repeated requests no report has been received.

Lamoille County

Report of election of officers received and printed in the Roster.

Northeastern County

This Society has held three meetings during the last year with an average attendance of seventeen. It has been nearly impossible to obtain speakers from outside of the District and the physicians in the district seem to be too busy to write papers for the meetings. The spirit in Caledonia County for medical meetings, I believe is very good if a program could be arranged for them. My suggestion would be instead of leaving this work for the Secretary, the President should appoint four members or more to arrange a meeting in a certain month and have at least four meetings a year. Then have them notify the Secretary of their program and he notify the members.

Willis B. Fitch, M.D., *Secretary.*

Rutland County

The first regular meeting was held January 16th. Drs. Howard Ball and John Southworth were elected to membership. Dr. Urquhart of the Connecticut State Sanitarium spoke on the Surgical Treatment of Tuberculosis. He demonstrated a phrenicectomy before the Society.

The next meeting was on April 15. Drs. H. B. Loder of Brandon and E. R. Mitiguy of Poultney were elected to membership. Dr. Frederic J. Cotton of Boston spoke on Osteomyelitis.

The next meeting was held July 9. This was the annual meeting and election of officers. The address of the evening was by Dr. Oswald S. Lowsley of New York, chief of the Urological Section of the

New York Hospital, whose subject was Newer Methods of Diagnosis and Treatment in Urologic Surgery. This meeting was preceded by a two-day clinic at the Rutland Hospital—an orthopedic and surgical clinic by Dr. Frederic J. Cotton; and a medical clinic by Dr. Richard Ohler of Boston. Dr. Robert Williams of Rutland was elected to membership at this meeting.

The average attendance at the meetings last year was twenty-eight.

At the next meeting to be held the last of October, Dr. Quick, Director of the Memorial Hospital in New York, will hold a cancer clinic in conjunction with the opening of the New Rutland Hospital.

B. F. Cook, M.D., *Secretary.*

Washington County

We are making great plans for our annual meeting which is to be held at Camp Randolph, Highgate Springs, Vt., the guests of Randolph physicians. Some are planning to stay overnight and try their luck at fishing for Big Ones. Our meetings have been held on different dates than has been the custom in the past. On account of the members being so widely separated from Richmond to Bradford and from Hardwick to Randolph, many are unable to attend because of traveling conditions, so we are trying to hold the meetings in May, July, September and November, the September meeting being called the annual meeting.

The May meeting was held at the Vermont State Hospital at Waterbury, where Dr. J. C. O'Neil, Assistant Superintendent, showed several cases of Huntington's Chorea. This was very interesting as Dr. O'Neil showed the sixth generation of a choreic family who have been patients at this Hospital. This meeting was well attended.

The second meeting (July) took the form of an outing at Lake Fairlee. This was arranged by Dr. L. B. Allen of Post Mills, who showed how Bean hole beans and hot dogs are supposed to be served. There was no business meeting at this time.

As before stated the September meeting will be held at Camp Randolph and the November meeting in Barre.

Washington County Society has lost three men in the past year in the death of Dr. W. B. Mayo, and the removal from the state of Dr. Fred Kalch of So. Strafford and Dr. E. O. Angel of Bradford. We have taken into membership Drs. R. H. Bisson, Montpelier, G. B. Goddard of Northfield, J. A. Dow of W. Topsham, G. A. Weaver of Bradford, and F. B. Jennings of Randolph, so that now we have 54 members including three honorary members.

I wish that something could be done to stimulate interest in our Society Meetings. I can tell before each meeting just who to expect to see, because it is always the same old reliables who make things worth while. Since becoming a member, now five years, I have failed to see some 15 or 20 men at any meeting, these men are not the most active ones by any means. If our meetings are void of interest, won't they do their bit to change things? Possibly the change in dates will have some effect, but so far there has been no change.

Charles H. Goyette, M.D., *Secretary.*

Windsor County

We have lost one member since the last annual meeting and added three new ones and there are two applications now pending. The activities during the past year have been entirely lacking, probably through the fault of the officers in not pushing things a little.

I wish we might each year have one meeting such as was held at Windsor a year ago last Spring, at which time the four talks were given on Infant and Maternal welfare, by members of the State Society and members of the State Board of Health, because such a program is enjoyable and profitable to all the members.

The only method by which a semi-annual meeting can be secured by each and every County Society in the State will be by a stated visitation of one or more of the State Society Officers, such as is made by the District Governor in Rotary. After all, the strength of the State Society is largely due to the interest stirred up in the County Societies.

A. B. Woodman, M.D., *Secretary.*

Windham County

We do not know whether we are having the annual meeting before the State meeting or not. We are planning on having a Clam-bake in a week or so to celebrate the 50th Anniversary of the Society and may have the annual meeting with this.

We have had only one meeting of the Society this year, on May 14, 1930, which was poorly attended, only eight members being present. At this meeting, three new members were taken in.

At our last meeting a motion was passed instructing the Secretary to write and inform you that Dr. Solomon, from Brattleboro, was located in Norwood, Mass. I also ask you to inform the Massachusetts State Secretary of his change of location. I would like to hear from you if you do this before our next meeting.

Am enclosing a letter from Mrs. Geo. D. Buxton. It seems to me that Dr. Buxton's dues ought to be refunded, as he was a good member of the Society. I would like you to give this your attention.

Sorry that I cannot give a report of more activities, this year.

John A. Stewart, M.D., *Secretary.*

REPORT OF THE PUBLICATION COMMITTEE

Such prepared papers as were given at the annual meeting 1929 and such County notes, Obituaries, Board of Health items and other matters as could be collected have been printed monthly in the Vermont issue of the *New England Journal of Medicine.*

During the last few months the material from New Hampshire and Vermont has appeared in the same issue, so that the members of the two states could follow the activities of our Society.

William G. Ricker, M.D., *Chairman.*

REPORT OF EXECUTIVE COMMITTEE

The Executive Committee, whose duty it is to prepare the program for the Annual Meeting, has submitted said program as their Annual report.

On account of illness in the family, the Chairman of the Committee was forced to leave home and the copy up to that time was given to the printer, hence the omission of names to discuss the various papers.

William G. Ricker, M.D., *Chairman.*

REPORT OF THE MEDICO-LEGAL COMMITTEE

To the Vermont State Medical Society:

During the past year your Medico-Legal Committee has had two new cases referred to it. Of these, one has been disposed of satisfactorily. The other is still pending.

At the beginning of the year one old case was remaining. During the year this has reached the court and been decided favorably to the Society.

An examination of the treasurer's report shows the expenses for the year small, thus leaving us a satisfactory reserve.

Respectfully,
Edwin A. Hyatt, *Secretary.*

REPORT OF NECROLOGY COMMITTEE

Following is the list of deaths of members of the Society during the past year. Their obituaries

have been published in the Vermont issue of the *New England Journal of Medicine*.

Dr. A. L. Miner, Bellows Falls, Nov. 9, 1929.
Dr. E. R. Lynch, Brattleboro, Jan. 16, 1930.
Dr. E. J. Fish, So. Royalton, Jan. 25, 1930.
Dr. G. D. Buxton, Cavendish, Feb. 13, 1930.
Dr. H. R. Watkins, Burlington, Feb. 19, 1930.
Dr. S. E. Darling, Hardwick, April 8, 1930.
Dr. E. T. Brown, Burlington, June 4, 1930.
Dr. Wm. B. Mayo, Northfield, April 25, 1930.
Dr. D. S. Drake, White River Jct., August 30, 1930.

L. A. Russlow,
B. D. Adams,
H. S. Pierce.

REPORT OF THE NEW ENGLAND MEDICAL COUNCIL

To the Vermont State Medical Society:

The New England Medical Council held its session last October, in conjunction with the annual meeting of the Vermont State Medical Society. Since then, a session has been held in Boston, which was well represented by each of the New England states.

At these meetings subjects which are of mutual concern to the New England section are discussed, and an attempt is made to improve conditions for all New England practitioners.

Full reports of these meetings are printed. If any member wishes additional copies they may be had by addressing any one of your members of the council.

Edwin A. Hyatt.

REPORT OF THE TREASURER

For the Year Ending September 12, 1930

General Account

Receipts

1929	
Sept. 12 Balance on hand	$1,322.04
Oct. 21 Recd. from Victor X-Ray Corp.	7.50
Oct. 21 Recd. from Surgeons & Physicians Supply Co.	15.00
Oct. 21 Recd. from Davies Rose & Co., Ltd.	15.00
Oct. 21 Recd. from Tailby-Nason Co.	15.00
Oct. 21 Recd. from Lederle Antitoxin Lab.	15.00
Nov. 4 Recd. from Vermont Peoples Nat. Bank, income Dr. H. D. Holton Trust Fund	65.00
During Fiscal Year	
Recd. from Addison Co. dues $5. each	50.00
Recd. from Bennington Co. dues $5. each	95.00
Recd. from Chittenden Co. dues $5. each	365.00
Recd. from Franklin Co. dues $5. each	125.00
Recd. from Lamoille Co. dues $5. each	40.00
Recd. from Rutland Co. dues $5. each	250.00
Recd. from Washington Co. dues $5 each	235.00
Recd. from Windham Co. dues $5. each	130.00
Recd. from Windsor Co. dues $5. each	105.00
Recd. from Northeastern Co. dues $5 each	165.00
Balance on hand and receipts	$3,014.54

General Accounts

Disbursements

1929	
Sept. 14 Paid The Lilley Co. badges	$24.41
Sept. 21 Paid Martha C. Blodgett, labor for Secretary	9.15
Sept. 28 Paid Dr. William G. Ricker, cash paid for stenographic work	$5.00
Sept. 28 Paid Passumpsic Sav. Bank, rent Secretary's safe deposit box	7.50
Oct. 1 Paid The Cowles Press, printing programs, annual meeting	34.30
Oct. 12 Paid Hotel Windham entertainment of guests of the society	72.80
Oct. 19 Paid Remington Rand, Business Service, special paper for Secretary	7.90
Oct. 19 Paid Dr. F. N. Sperry, expenses	10.50
Oct. 21 Paid Dr. F. G. Blake, expenses, Drs. Trask, Van Allen and Blake	34.15
Oct. 21 Paid Dr. J. S. Hill, stamped envelopes	1.00
Oct. 21 Paid Lucile J. Parker, stenographic work	1.50
Oct. 21 Paid James' Studio, negative and prints of Woman's Club Rooms	10.00
Oct. 23 Paid Dr. J. N. Jenne, part expenses, delegate to A. M. A., Portland, Oregon	100.00
Oct. 23 Paid Dr. Benjamin White, income Dr. H. D. Holton trust fund	65.00
Nov. 2 Paid *New England Journal of Medicine*, reprints, envelopes and postage	84.23
Nov. 2 Paid Fred Exner, orchestra at banquet	25.00
Nov. 2 Paid Mrs. M. D. McAuliff, rent of Woman's Club Rooms	14.00
Nov. 2 Paid L. S. Hayes, services registration clerk	5.00
Nov. 2 Paid The Windham Press, printing menus	4.75
Nov. 2 Paid Dr. J. H. Blodgett, expense, telephones, etc.	5.90
Nov. 2 Paid Dawn Nelson Wallace, entertainment	16.00
Nov. 2 Paid Dr. Frederick W. Bancroft, expenses	30.00
Nov. 23 Paid *New England Journal of Medicine*, Journal for July, Aug. and Sept.	108.56
Nov. 23 Paid Dr. William G. Ricker, salary and expenses	129.42
Nov. 30 Paid *New England Journal of Medicine*, reprints	1.17
Dec. 7 Paid *New England Journal of Medicine*, one half cost of photograph of Dr. Geo. R. Anderson	1.56
Dec. 14 Paid Susan A. Nott, reporting annual meeting and transcript	121.87
Dec. 31 Paid T. S. Peck Insurance Agency, Treasurer's Bond	12.50
Dec. 31 Paid *New England Journal of Medicine*, reprints	5.27
Dec. 31 Paid Dr. Walter P. Bowers, one sixth expenses, New England Medical Council	29.97
Dec. 30 Paid *New England Journal of Medicine*, Journal for Oct., Nov., and Dec.	113.04
Feb. 15 Paid *New England Journal of Medicine*, reprints	7.50
Feb. 15 Paid The Cowles Press, printing statements	5.00
Mar. 5 Paid *New England Journal of Medicine*, cuts for printing	34.65
Apr. 12 Paid *New England Journal of Medicine*, Journal for Jan., Feb., and Mar.	114.59
Apr. 14 Paid New England Medical Council, our share of expenses	55.31
May 7 Paid *New England Journal of Medicine*, subscription Mrs. A. L. Miner for 1930	1.00
May 7 Paid The Cowles Press, letter heads	18.62
Sept. 11 Paid *New England Journal of Medicine*, subscription Mrs. A. L. Miner, for 1931	1.00
Sept. 11 Paid *New England Journal of Medicine*, Journals for Apr. and May	77.76

Sept. 12 Paid David Marvin, postage and telephones	$6.50
Total Disbursements	$1,413.38
Balance on hand	1,601.16
Total disbursements and funds on hand	$3,014.54

MEDICO-LEGAL ACCOUNT

Receipts

1929	
Sept. 12 Balance on hand	$11,399.04
1930	
Jan. 1 Recd. interest on savings account	210.63
July 1 Recd. interest on savings account	218.18
During Fiscal Year	
Recd. from Addison Co. dues $5. each	50.00
Recd. from Bennington Co. dues $5. each	95.00
Recd. from Chittenden Co. dues $5. each	330.00
Recd. from Franklin Co. dues $5. each	125.00
Recd. from Lamoille Co. dues $5. each	40.00
Recd. from Rutland Co. dues $5. each	250.00
Recd. from Washington Co. dues $5. each	230.00
Recd. from Windham Co. dues $5. each	130.00
Recd. from Windsor Co. dues $5. each	105.00
Recd. from Northeastern Co. dues $5 each	165.00
Balance on hand and receipts	$13,347.85

Eight members exempt from dues

Disbursements

1929	
Sept. 23 Paid Dr. E. A. Hyatt services and expenses, Medico-Legal Com.	$35.45
Total disbursements	35.45
Balance on hand	13,312.40
Balance on hand and disbursements	$13,347.85

TOTAL ASSETS

General Account	$1,601.16
Medico-Legal Fund	13,312.40
Total assets	$14,913.56

Respectfully submitted,
DAVID MARVIN, *Treasurer.*

REPORT OF THE AUDITOR

I have examined the books of our Treasurer, Doctor David Marvin, and have the pleasure to report that I find them correct.

GEORGE M. SABIN, *Auditor.*

September 22, 1930.

NEWS ITEMS

BENNINGTON COUNTY MEDICAL SOCIETY

The Bennington County Medical Society met September 29 at the Putnam Hospital, Bennington, Vt.

The matters of examination of school children and the administration of diphtheria toxin-antitoxin to children in schools received much attention. The consensus of opinion was that physicians should be paid a fair price for these services. The dentists charge $10.00 per hour for their services in the schools.

If a dentist is worth $10.00 per hour a physician should be worth as much.

Dr. E. A. Tobin showed a series of very interesting x-ray plates which depicted a case of transposed viscera. The first knowledge of this case came when considerable difficulty was experienced in finding the appendix during operation. It was finally located on the left side.

Dr. Trotter demonstrated a case of fracture of the right femur in a boy of 3.

Dr. Ellis Kellert demonstrated a case of rupture of the heart. Examination of the heart revealed a small rupture in the right auricle. The deceased had apparently been in good health up to the time he was stricken.

The regular meeting of the Bennington County Medical Society was held Wednesday, October 29, 1930, at the Putnam Memorial Hospital.

Dr. Ellis Kellert showed a number of very interesting gross pathologic specimens recently removed.

The next exhibit was a brain containing a glioma. The tumor was in the right parietal region and was spherical and about 1¼ inches in diameter. The third specimen was a fresh brain containing a large blood clot and a cerebellum which contained several small tumors.

Dr. Frank E. Dean demonstrated several hospital staff cases, one a case of partial paralysis of right forearm following an injury to the humerus. The patient, a young woman, fell injuring the head of the humerus possibly fracturing it, the x-ray evidence not being conclusive; paralysis supervened.

Dr. L. H. Ross demonstrated a case of atypical tuberculosis in a man of 26.

The annual election was held and the following chosen:

President: E. M. Gardenier.
Vice-President: H. W. Elliott.
Secretary-Treasurer: J. C. Armstrong.
Auditor: Edw. A. Tobin.
Delegate: C. S. Buchanan.
Alternate: J. D. Lane.
Delegate: L. H. Ross.
Alternate: E. A. Tobin.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 16491

ACUTE FEVER AND PRECORDIAL PAIN

MEDICAL DEPARTMENT

An American clerk twenty-four years old entered September 11 in semicoma. The history was given by his wife. The chief complaints were coma and fever.

Beginning two weeks before admission he had steady pain over the right eye for several days. A physician found his temperature 103° at the onset. The patient stayed in bed for several days, feeling miserable. Ten days before admission he went to Maine for the week-end, still with right frontal pain which did not last for such long periods and wandered more or less over his head. He spat white mucus which apparently came from the nose and mouth. He seemed restless and feverish. Nine days and eight days before admission he felt fairly well, but was still weak and without appetite. Seven days ago his temperature was 104°. During the next few days the sputum became blood-streaked. Six days ago he had aching all over. The following day his physician heard a few squeaks in his chest. His temperature was 102°. Three days before admission he seemed a little better, with a temperature of 100°. The next day there was no congestion in the lung. He had a temperature of 101° and a little tickling cough with blood-streaked sputum. He began to seem a little dazed and his speech was slow. The day before admission his temperature was normal and he again complained of a dull ache over the right eye and an ache in the precordial region with occasional sharp catches on taking breath. The morning of admission his wife was awakened by his tossing his head from side to side, more to the left. Ten minutes later he began thrashing about and fighting to get out of bed, so that it took four persons to hold him. His eyes were rolled outward. He moaned and tried to bite his wife's hands. The attack lasted three-quarters of an hour. At five o'clock he was quiet and dazed but responded to questions. He was given a hypodermic and slept four hours. At ten o'clock in the morning the temperature was normal, the patient rational. At eleven o'clock he had another attack of restlessness and was sent to the hospital.

The family history is not significant. He had been married three months.

His wife knew of no serious illnesses in the past. He had had several carbuncles and felons recently. He had a feeling of fullness and distress after meals, with some burning. His habits were good.

Clinical examination showed a fairly well nourished man in semicoma, drooling, not responding to tactile stimuli or questions, but occasionally opening his eyes and looking about in a dazed manner. The skin was hot, with profuse perspiration. The chin could not be brought to the chest. The neck moved freely in other directions. The left border of cardiac dullness was 9 centimeters to the left of midsternum, one centimeter outside the midclavicular line. The aortic second sound was greater than the pulmonic second. A soft systolic murmur was heard at the base. The pulses were normal, the blood pressure 140/40. There was a loud leathery friction rub over the right base extending half way up the back and into the axilla. There were a few moderate crackles at the left base. The abdomen was normal. The pupils were fixed to light. Both fundi showed engorged veins. The right nasal disc margin was slightly blurred, perhaps within normal limits. No hemorrhage or exudate was seen. The epigastric, abdominal, and cremasteric reflexes were absent. The knee jerks were active, variable, the left occasionally greater than the right. Babinski was variable, occasionally positive, generally negative. There was variable pseudoclonus of both ankles. Kernig was negative.

The amount of urine was normal when recorded, the specific gravity 1.020 to 1.022, the urine cloudy at all of three examinations, showed the slightest possible trace of albumin at two, leukocytes in two specimens, rare red cells in one, bacteria in all. The blood showed 32,400 to 26,500 leukocytes, 88 to 89 per cent polynuclears, hemoglobin 75 per cent, reds 4,400,000, slight variation in shape, slight achromia, platelets fairly normal in one smear, increased in another. Hinton negative. Sputum negative for tuberculosis. The findings in five lumbar punctures are shown in the table.

SPINAL FLUIDS

Date	Amount	Character	Pressure		Cells		Total protein	Colloidal gold	Sugar	Ammonium sulphate
			Init.	Final	Total	Poly.				
Sep. 10	15 c.c.	Clear colorless non-clotting	270	140	16	60%	51	1111233210	86	+++
Sep. 11	20 "	" " "	400	150	11	23%	53	1222332100	84	++
Sep. 12	20 "	" " "	250	80	5	—	57	1112233221	81	0
Sep. 15	20 "	" " "	450	60	50	43%	52	—	75	++
Sep. 16	15 "	" " "	200	80	166	83%	—	—	—	0

Wassermann tests were negative on September 10 and 12

X-ray examination showed the right side of the diaphragm elevated, sharp in outline. The left side was normal. The interlobar pleura on the right side was visible. Both lung fields were slightly less radiant than normal. There was diffuse thickening of the larger lung markings. There were multiple indefinite areas of dullness in the left side. The apex and the second and fourth interspaces showed these areas best. The variations from normal were not sufficient to make a positive statement possible. There was a suggestion, however, of multiple areas of beginning consolidation and pleurisy on the right side. Two days later the sinuses were not definitely abnormal. The appearance of the chest had changed. The right diaphragm was still high, and there was a band of dullness along the right axillary line about half an inch in width. At the right base was dullness of a fine mottled character and the interlobar pleura was visible. There was a sharply defined rounded area of density in the left third interspace which measured two centimeters in diameter. All the lung markings were diffusely thickened and beaded.

The temperature was 99.5° to 104°, rectal, the pulse 62 to 137, the respiration 20 to 52.

Soon after admission the patient began to have convulsive movements of the left arm with the hand drawn tightly in the position of carpal spasm. At the same time the head and eyes were drawn to the left, with twitching of the left facial muscles. The tongue and legs did not move. The convulsions lasted about ten minutes. In the course of the next hour he had several similar attacks, but varying in the areas involved,—winking of the right eye alone, twitching of the muscles of the right side of the chin, drawing of the head and eyes to the right with twitching of the right side of the face and the left leg; but the predominant type was clonic movements of the left arm. Nystagmus of an impure rotatory type was observed at times.

Lumbar puncture was done about six p. m. During the evening the patient had an occasional convulsive attack lasting a few minutes, always involving the left arm. At midnight he was considerably brighter, responding to simple questions, but extremely restless. Morphia grain one-fourth was given subcutaneously twice during the night without marked effect. The following morning he was still very restless and had clonic movements of the left arm, but was much more responsive. The pleural rub at the right base had increased considerably in extent.

On September 11 a neurosurgical consultant found no evidence of a surgical condition. He noted a tendency towards left ankle clonus and Babinski.

September 12, after a quiet night on two doses of morphia with hyoscin, the patient had a left sided convulsion lasting three minutes, starting in the face and extending to the arm and hand. He seemed brighter, asked for food, and had a comfortable night. September 13 there was a loud rub in the left axilla. The tongue was red, the throat beefy. The right knee jerk was greater than the left. There was left clonus and Babinski. In the afternoon the patient complained of seeing double on looking to the right.

On September 14 he seemed duller, had thick speech, repeated words over and over, and moaned almost constantly. Examination showed slight internal squint, whether of one eye or both could not be determined. There was one white patch adherent to the left anterior pillar of the throat, with several smaller pinpoint ones, chiefly on the left side. No rub was heard in the left chest. A faint rub remained in the right axilla, with dullness and diminished spoken voice. There were a few squeaks and moist râles at the right base. September 15 there was weakness of the left external rectus muscle. He protruded his tongue in the midline on request, though he did not answer questions. The left arm was held spastic; no voluntary motion or reflexes of the arm could be elicited. The left leg was also spastic, and there was positive Kernig and definite clonus on the left. The rub at the right base was much diminished, but there was dullness, probably a small amount of fluid as shown by x-ray. The breath sounds in the right front chest to the level of the third and fourth ribs were almost bronchial. The patient looked very toxic and voided involuntarily. A nerve consultant reported, "I have seen this man three times. . . . Now he exhibits a well marked left spastic condition of the arm and leg, but also shows right Babinski. Mentally, cerebration is almost at a standstill. The cranial nerves are now involved, a new sign—the left sixth and probably palatal palsy. The discs have gradually become more blurred and now I think are slightly elevated. . . . I cannot see any indication at present for cranial operation. I advise daily lumbar punctures."

That night the patient had three convulsions after the lumbar puncture. September 16 he was much worse, unconscious and hiccuping. The right disc was considerably more blurred than the day before. The left fundus could not be seen. The respirations were noisy and rapid. He had much froth in the mouth. There were extremely loud pleural rubs in both chests in front. The left arm was now relaxed and the right arm spastic. During that evening he stopped breathing for a few seconds twice. At midnight the respiratory rate suddenly increased, the patient seemed to choke, had bleeding from the nose, became deeply cyanotic, and died.

Clinical Discussion

BY RICHARD C. CABOT, M.D., AND GEORGE W. HOLMES, M.D.

NOTES ON THE HISTORY

That is a very queer history. First we think of a frontal sinus, then we think of pneumonia, then of meninigitis. It does not fit very well with any of them.

NOTES ON THE PHYSICAL EXAMINATION

There was retraction of the neck and presumably stiffness in it.

The negative Kernig is very surprising.

All the spinal fluids are clear, show no clotting, and all show an increase of cells. This would make one suspect meningitis or some local brain abscess. The sugar is not particularly high. Those facts point to meningitis of a septic type, perhaps coming from the sinus or from some other cavity near the brain or as an epidemic. Of course the pressures are notably high.

This is a urine that goes with fever.

The blood shows an increase in polynuclears, as one would expect in meningitis.

The multiple areas of dullness seen by x-ray were on the side on which the diaphragm was normal, but I should suppose he had evidence of bronchopneumonia in both lungs.

Dr. George W. Holmes: The signs on the right are definitely those of pneumonia and pleurisy. Ordinarily they would mean lobar pneumonia. The high position of the diaphragm probably means that on account of the pleurisy and pain the patient did not take a deep breath. The films were taken at full inspiration. The area of density on the left is hard to explain. It may be an old lesion in the lung of no present importance. That rounded, sharply defined dense area is a little suggestive of metastatic malignancy. It could also be a bronchopneumonic patch. The thickened and beaded lung markings mean nothing more than infection in the lung.

DIFFERENTIAL DIAGNOSIS

Dr. Cabot: I should like to start from the best accredited fact, which is the fact of meningitis. We have the evidence of irritation in the meninges proved by the spinal punctures. There is considerable evidence in other parts of the body to make us suspect meningitis. We have no definite evidence against the heart or any abdominal organ. Hence we should concentrate our attention on the brain and on the lungs. At the beginning the symptoms in the brain are not localized at all. Later we are looking for trouble on the right side of the brain more than elsewhere. What trouble could that be? I suppose it could be a one-sided internal hydrocephalus, though I have never seen one. The other probability is brain abscess, which would associate itself with abscesses in the lung. He has had carbuncles, that is he has had a staphylococcus lesion. That could involve the lung and the brain. That seems the best diagnosis I can make,—multiple abscesses of the lung and brain, accounting for the pleurisy and the cerebral lesions of meningitis, for the localized symptoms and for the spinal fluid as part of such an abscess process. The frontal sinus was another manifestation I think of the general staphylococcus sepsis to which the lung abscesses and the brain abscess were due. Abscess of the heart wall itself often goes with such lesions as this. If he did have abscesses of the lung and the brain there is a good chance he had abscess of the heart wall too. Abscess of the kidneys might also go with staphylococcus infection.

Suppose it was not an abscess at all. What else could it be? Bronchopneumonia with septic meningitis and internal hydrocephalus. But I am rather bothered by that's being unilateral. Cerebral tumor is to be considered. But I do not see how it could cause this spinal fluid. I have never seen so much spinal fluid with a cerebral tumor. We can have cerebral tumor at twenty-four, but tumor does not often occur in the lung with metastasis in the brain at twenty-four. Brain tumors are generally not metastatic, but primary.

A Student: How would the picture of encephalitis differ from this?

Dr. Cabot: In the first place he did not begin with diplopia. Then drowsiness is not on the whole characteristic of this case. There is very little tendency to generalized choreiform movements. There are more spasms and more convulsions than I should expect with encephalitis. There is a higher leukocyte count, and there are more lung signs. I am willing to vote against it.

A Student: How about tuberculous meningitis?

Dr. Cabot: Tuberculous meningitis does not properly give such a fluid in the spinal canal. It ought to give a lymphocytic rather than a polynuclear fluid. It is not ordinarily associated with this sort of lung lesion. It does not usually have such convulsions. We have not seen those of course; we have only heard them described. It does not sound like miliary tuberculosis, which is usually the form of tuberculosis associated with tuberculous meningitis. In that moreover there is not usually such a high count of blood leukocytes. I am willing to vote against that.

A Student: Do you think the spinal fluid sugar would be lowered in a case of meningitis, or isn't that an important factor?

Dr. Cabot: I do not think I am able to answer that question. I should say yes, usually, but that it is not so constant that we should place great stress on its absence. Certainly the blood sugar is not so high as in sleeping sickness.

A STUDENT: Aren't the bacteria in the urine significant?

DR. CABOT: I should not suppose so, unless we know how long it stood before being examined.

A STUDENT: Isn't the cell count lower than it usually is in encephalitis?

DR. CABOT: Yes, I should say it is. I think this is probably not a purulent process in the spinal canal but merely an irritation that goes with it.

A STUDENT: Do you get these periods of quietness in septic meningitis such as he has?

DR. CABOT: Yes.

A STUDENT: Were cultures done on the spinal fluids?

MISS PAINTER: No.

CLINICAL DIAGNOSES

Tuberculous meningitis.
Acute miliary tuberculosis.

DR. RICHARD C. CABOT'S DIAGNOSES

Staphylococcus sepsis.
Multiple staphylococcus abscesses in the lung and brain.
Septic meningitis.
Septic sinusitis.
Possibly abscesses of the heart wall and the kidneys.
Pleurisy.

ANATOMIC DIAGNOSES

Septicemia, staphylococcus aureus.
Septic meningitis.
Multiple abscesses of the lung.
Acute endocarditis of the mitral valve.
Acute frontal sinusitis.

PATHOLOGIC DISCUSSION

DR. MALLORY: Autopsy showed meningitis and multiple abscesses in the lungs with acute endocarditis of the mitral valve. There was also an acute frontal sinusitis. There was no demonstrable connection between that and the meningeal involvement. Sometimes the process can be shown to extend directly to the meninges by perforation of the posterior wall of the sinus, or more commonly the infection spreads in the form of a septic thrombosis of the longitudinal sinus. Neither of these was found in this case. I still think it is fairly probable that the sinusitis was the primary cause of the trouble, and that early drainage might have prevented the subsequent complications. There was rather a patchy type of meningitis scattered over the brain.

Cultures from the spinal fluid, the lungs and the heart all showed staphylococcus aureus.

DR. CABOT: Why did he have so much in the way of left-sided symptoms?

DR. MALLORY: We do not feel that we have any satisfactory explanation of that.

DR. CABOT: He had no hydrocephalus?

DR. MALLORY: No.

CASE 16492

ONE YEAR'S INTERMITTENT EPIGASTRIC PAIN

MEDICAL DEPARTMENT

A Canadian woman forty-two years old, an attendant nurse, entered May 26 complaining of intermittent pain and soreness in the pit of the stomach of a year's duration.

Since the onset she had had five or six attacks similar to the present one lasting from one to three days. The present attack began eight days before admission, with sharply localized epigastric pain beginning half an hour after luncheon, gradually increasing in severity and passing off somewhat later. For three days she had this pain off and on, with no apparent relation to meals. For the past five days it had been constant, although it varied somewhat in intensity. It was accompanied by cutaneous soreness. When it was worst it went straight through to the middle of the back and extended a short distance along the costal margin on each side in front. When most severe it was griping and her stomach felt distended. She had stayed in bed for the past five days because the pain was less when she was quiet. Nothing gave relief except a hot water bottle and soda. The latter she took in such quantities that it caused vomiting. In the past five days she had taken little except rice, cream and weak tea. On two mornings she had felt slightly nauseated, but had had no vomiting except when she induced it. During the past few months her weight had fallen from 127 pounds to 116 and she had lost appetite. She thought there had been some mucus in her stools during the past few days.

The family history is not significant.

At eighteen she had an attack of rheumatic fever. At twenty she had three severe hemorrhages from the lungs and her sputum was repeatedly positive for tuberculosis. She spent a year and a half at Saranac. She had been "cured" for four years. Sixteen years before admission, after six months of indigestion and a severe attack of pain, her gall bladder was drained. Six months later it was removed. Ten years before admission her tonsils and adenoids were removed because she had many colds and sore throats. She had had no sore throats since that time. Nine years before admission her appendix was removed. About that time she began to notice increasing dyspnea on exertion, slight non-productive cough, and frequent palpitation even without exertion. For the past three months she had been taking digitalis. For six months her menstrual flow had been less. The

last three periods had come at intervals of fourteen to eighteen days.

Clinical examination showed a well-nourished woman lying in bed, quietly complaining of pain. The skin was lightly sallow, the mucous membranes pale. There was visible arterial pulsation in the neck and a palpable impulse in the suprasternal notch. There were scars of old tuberculous glands on the right side. The apex impulse of the heart was felt in the fifth space 10.5 centimeters to the left of midsternum and one centimeter to the left of the midclavicular line. The left border of dullness was 11 centimeters to the left. The rhythm was regular, the action strong. There was a short soft murmur prolonging the first sound, best heard at the base. The pulmonic second sound was distinct. The aortic second sound was replaced by a harsh blowing murmur extending into diastole but starting before the pulmonic second was heard. This was heard all over the precordia, best in the aortic area. The blood pressure was 160/20. There was Corrigan pulse. Pistol shot was heard over the arteries. The upper half of the abdomen was full, somewhat tense and very tender. Deep pressure in the right flank caused pain in the midepigastrium and also to a much less degree in the left flank. The tenderness was so great that deep palpation was impossible and percussion could be done only very lightly. The area seemed somewhat tympanitic. It pulsated with the heart. There was slight tenderness in both costovertebral angles, more on the left. There was slight spasm of the left erector spinae. All the deep reflexes were very active. Bilateral patella jerks. Pupils normal. The fundi showed some tortuosity of the vessels and some silver wire appearance.

Urine: amount not recorded, specific gravity 1.024 to 1.016, a slight trace of albumin at one of two examinations, leukocytes at both. Blood: 8,500 to 15,500 leukocytes, hemoglobin 55 to 60 per cent, polynuclears 74 per cent, reds 3,100,000 to 4,040,000, moderate achromia, moderate variation in shape and size, platelets decreased. No Hinton or Wassermann recorded. Sugar 95 milligrams. Fasting contents of the stomach: 35 cubic centimeters of yellow material, no free hydrochloric acid, total acidity 4. Test meal half an hour later: 20 cubic centimeters, free hydrochloric acid 20, total acidity 26. Test meal one hour later: 23 cubic centimeters with mucus, free hydrochloric acid 36, total acidity 51. Guaiac negative on all three specimens. Stool clay colored, guaiac negative. Vomitus, guaiac negative. Icteric index 2 to 5. Quantitative and qualitative van den Bergh indirect reaction 0.3 milligrams per 100 cubic centimeters. Rosenthal liver function test, no retention of dye in 30 minutes.

Temperature 97° to 100°. Pulse 85 to 120, with a terminal drop to 77. Respirations normal except for a terminal increase to 28.

X-ray examination of the stomach without a motor meal showed the stomach apparently normal except for vigorous peristalsis. The first portion of the duodenum was constantly irregular and there was a considerable degree of spasm about the irregularity. Barium passed through the pyloric valve and duodenum readily. At a second examination a week later the duodenum was considerably less irritable than at the first examination and could be well filled. The only deformity was apparently due to pressure from the liver. There was no evidence of ulcer. The variations from the normal might have been due to periduodenitis. At twenty-four hours there was a considerable quantity of barium retained in the proximal half of the colon. There was a questionable irregularity in the left extremity of the transverse colon which it was thought should be checked by a barium enema. A film of the abdomen centered over the region of the pancreas showed no visible areas of calcification. No stones were seen. Films of the kidney regions and the dorsal and upper lumbar region were essentially negative.

In the ward the patient had complained of less pain, although morphia was often necessary. The pain seemed to reach its maximum in the late afternoon and evening without much relation to food. By June 8 she had lost eight pounds. She persisted in vomiting milk. The interne noted at the fluoroscopy that the heart shadow was triangular, with enlarged left ventricle and prominence in the region of the pulmonary conus, which with the auricles entirely obscured the shadow of the aortic arch. In the lateral view the left auricle was seen greatly enlarged and projecting posteriorly, where it displaced the esophagus to the left.

Additional history was now obtained from the patient's family. Since her cholecystectomy four years before her admission she had had attacks of abdominal pain or vomiting every month or two. The attacks commenced with pain and epigastric distention which continued for a day or two. At the end of that time she vomited large amounts of green or yellow material and in six to twelve hours had complete relief and could eat a large meal without discomfort. Toward the end of the attacks her skin was yellow. This cleared up after the vomiting. They insisted that there was no psychic element in her condition and that she had always borne extreme pain before calling for relief.

June 11 a surgical consultant found the abdomen generally full and tender, the greatest tenderness in the left upper quadrant. No masses were felt, but the examination was unsatisfactory. June 18 an examination was made under spinal anesthesia. Skin anesthesia was complete to the third rib anteriorly with motor paralysis of the lower extremities. The epigastric tenderness still remained as before. The patient could localize the pressure with extreme

accuracy. There was an indefinite feeling of something in the epigastrium. During the afternoon the patient was pale, sweating, and slightly cyanotic and complained of much pain and later of severe headache which continued the next morning.

A friend now gave the further history that the patient's attacks had followed one to three day drinking bouts. She had been an excessive user of alcohol for many years until a few months before admission. It was now observed that there had been only five days in the hospital when she had not had some opium, and on four of these she had had fever.

The visiting physician strongly favored operation, and a medical consultant thought the patient able to stand it. The first surgical consultant also advised it. The senior surgeon thought there was nothing to gain from it and possibly everything to lose. The patient was determined to be operated upon, and by the second surgeon. She continued to have severe pain.

June 25 operation was done. The following day the patient died.

Clinical Discussion

BY RICHARD C. CABOT, M.D., AND
GEORGE W. HOLMES, M.D.

NOTES ON THE HISTORY

I suppose if we read only the opening paragraph we should say peptic ulcer. But we should certainly have to add that peptic ulcer would never last only three days. It would last longer.

By a "griping" pain I suppose we generally mean a pain that comes with peristaltic rhythm. If it comes at regular intervals it would seem to be related to the movements of the gastro-intestinal tract. So I suppose this pain was intermittent at regular intervals.

As one reads along this gradually changes from a gastric to a cardiac history. But it is rather hard to say how that could give this sort of symptoms. Of course gall bladder adhesions could give some sort of symptoms. I do not know that they can be altogether ruled out. But she has at present more cardiac than gastro-intestinal or gall bladder symptoms.

Of course the tuberculous episode is important, because tuberculosis is one of the important causes of dyspepsia and indigestion of various kinds. It does not seem to me, however, as if tuberculosis could be causing this. At the present time, previous to reading the physical examination, I am looking to cardiac disease as the probable cause of her symptoms.

NOTES ON THE PHYSICAL EXAMINATION

The pulse pressure, if the record is correct, and I have no reason to doubt it, would go very far to give us a diagnosis of aortic regurgitation.

It seems to me we have every reason to suppose that the tenderness in the right flank is due to the liver,—congestion and pain because of it.

I think costovertebral tenderness is a term that means very little. You will find that you can produce it in almost any normal person. The tendon reflexes were a little more active than usual, I take it.

There is no good evidence of renal trouble so far as the urine shows it.

There is secondary anemia which we must somehow account for and which aortic regurgitation in itself will not account for.

The test meal shows low acidity but no achylia. I do not think we have any good evidence against the stomach.

It is rather surprising that the stool should be clay-colored, there being no jaundice.

With a negative liver function test, we have nothing in particular to say against the liver.

Dr. Holmes: In the examinations of the gastro-intestinal tract the fluoroscopic observations are usually much more important than the films. We have here a series of films taken to show the spine. The only thing that I can see that is at all unusual is that there are some small white spots which I should suppose were calcifications in the costovertebral region. You can also see a calcified area in the anterior view between the ninth and the tenth dorsal vertebrae. Such a localized area of calcification is usually due to some trauma with injury to the disc. It may have no bearing on the present condition.

Dr. Cabot: Are those calcifications ever of any importance, Dr. Holmes?

Dr. Holmes: They might occur in tumor, but we should not be able to make a diagnosis of tumor from the presence of calcifications. I think they are important in deciding whether the patient has had an injury. They would not be positive evidence. We have quite a series of films taken of the stomach. You see the normal gas bubble in the fundus of the stomach, the mid-portion and the duodenum. The peristaltic waves are fairly well shown as they pass over the stomach. I should think from those films that the stomach and duodenum are normal. Of course we are not justified in making such a statement from the films alone. In this other film of the stomach the duodenum is better shown. It is smooth and of normal shape. It is hardly possible that there could be an ulcer of any size present. The man who did the fluoroscopic examination was evidently a little uncertain about the colon. From these films I do not see anything that would lead to a diagnosis of a pathologic process.

There is here in the right iliac fossa a contracted area that is not inconsistent with carcinoma, but there is no evidence that that is constant.

I think if there were a new growth producing such a deformity as we see there the man who did the fluoroscopy would have no trouble in finding it, and there would be obstruction with a growth of that kind. We have none of those things here. I should call it a negative examination.

DR. CABOT: Up to date we have first-rate evidence of aortic regurgitation, pretty good evidence of congested liver and not much evidence of anything else. Doesn't that fluoroscopic note show a very unusual state of things, Dr. Holmes?

DR. HOLMES: I think it is merely stated in an unusual way.

DR. CABOT: What about that displacement of the esophagus?

DR. HOLMES: That happens occasionally. Whenever the left auricle is large it displaces the esophagus. I think that is what we should expect.

DR. CABOT: Then the evidence is not merely of enlargement of the left ventricle but also of enlargement of the left auricle, which I should not have predicted.

Whether that was jaundice or just sallowness that she showed at the end of her attacks we cannot tell.

We see that they had evidently been wondering whether there was a considerable psychic element in this patient's complaints. They tried to get evidence in confirmation of that and did not get it.

DIFFERENTIAL DIAGNOSIS

Before we know what the operation showed we have to say what the preoperative diagnosis would be. I am very much in doubt. I do not believe aortic regurgitation would account for all this pain. But what does account for it? I cannot name anything. Certainly I do not believe it is the stomach. I do not believe such continued pain as this without any particular relation to meals ever comes from a gastric ulcer, and the x-ray would rule that out. The gall bladder? There are a number of things that make us think of that. But I do not know how we can have pain of this kind with the gall bladder removed. My guess is that adhesions about the gall bladder was what the surgeon thought. I think he operated for something in the gall bladder or something due to it, very possibly adhesions. Stone in the pancreas the x-ray does not help us to rule out, does it, Dr. Holmes? One clay-colored stool is not evidence of trouble in the pancreas. Stone in the pancreas is a very hard diagnosis to make. I do not think I ever made it right. However, I do not know how to exclude it. We have no reason to blame the spine. Dr. Holmes is evidence against that. Her drinking habits might suggest cirrhosis. I do not know any other disease of the liver that would give us such symptoms. But there is no portal obstruction. Syphilis of the liver she might have, but that is not a painful disease. Malignant disease of the liver or of the bile ducts? Very possibly: but we have no evidence beyond pain, and it is very dangerous to make a diagnosis on pain alone.

As you remember, one surgeon did not think there was anything to operate upon. "Adhesions or nothing," he thought. I imagine that was the reasoning of the first surgeon. Another said, "This patient wants an operation. We do not know much about the diagnosis, but there may be something to remove." I should say it was an operation done for exploration with the possibility of adhesions. Are there any other suggestions from the class before we look?

A STUDENT: Would the history of sixteen years of adhesions give you that?

DR. CABOT: I think not, but I do not know much about adhesions.

A STUDENT: How about gastric crises of syphilis?

DR. CABOT: That is a good idea. So far as I know the symptoms would be right, except that they are a little too continuous. We have no other evidence that I know of for syphilis except aortic regurgitation. We have no Wassermann report. I do not know why. I cannot exclude syphilis. As you know, I am always slow to make a diagnosis of syphilis without compelling evidence. We have no evidence of it in the nervous system. We may have evidence of it in the heart. If it is that in the heart the pain perfectly well could be gastric crises so far as I know. It is about as good a guess as anything else.

A STUDENT: Could there still be a stone in the common duct that was overlooked at the operation?

DR. CABOT: I do not think I know enough surgery to answer that certainly. But it does not seem probable. Stone in the common duct usually produces jaundice and fever. She does not have those. She did have some fever while here. There was no history of it before.

A STUDENT: Couldn't her gastric symptoms be accounted for by cirrhosis of the liver?

DR. CABOT: No, I think not. I think we do not ordinarily get gastric symptoms in cirrhosis of the liver.

A STUDENT: Could the gastric symptoms be caused by alcoholism,—alcoholic gastritis?

DR. CABOT: Gastritis is a very vague term. But I do not think that can account for her pain. I am troubled about that. I do not believe that it is merely passive congestion of the liver or that it is merely psychic.

A STUDENT: Could there be an aneurysm of the abdominal aorta?

DR. CABOT: I think not. Of course with syphilis aneurysm should be considered. But abdominal aneurysm could ordinarily be felt, and they gave a spinal anesthetic, so that they ought to have felt it.

A STUDENT: Might not that have been a mitral stenosis?

DR. CABOT: Of course the big left auricle suggests that.

A STUDENT: She had a previous history of rheumatic fever. That would give her gastric pain, wouldn't it?

DR. CABOT: I think not. We still have nothing to account for this pain.

A STUDENT: Could she get aortic regurgitation from rheumatic heart disease?

DR. CABOT: Certainly. I believe it is that. My belief is against syphilitic aortic regurgitation.

A STUDENT: Is that an aneurysm that pulsates in the neck?

DR. CABOT: No; such pulsation is very common in aortic regurgitation.

A STUDENT: Are you ruling out tuberculosis altogether?

DR. CABOT: I have not thought much about abdominal tuberculosis. It is not ordinarily a painful disease.

A STUDENT: Would the x-ray rule out tuberculous cold abscess?

DR. HOLMES: I think so, in an adult. In a child sometimes we find an abscess without any bone destruction or deformity, but I have never seen it in an adult.

A STUDENT: Could it be carcinoma of the duodenum?

DR. HOLMES: I do not believe so. We ought to have a great deal of deformity with carcinoma of the duodenum. It is extremely rare.

A STUDENT: Is carcinoma of the pancreas painful?

DR. CABOT: Yes, ordinarily.

PREOPERATIVE DIAGNOSIS

Localized peritonitis.
Possible perforation of the stomach.

OPERATION

Under ether a rounded resistance was felt in the epigastrium. The abdomen was opened with marked edema of the peritoneum. There was some free fluid lying over a mass of adhesions between the duodenum and the liver. The right lobe of the liver was felt, engorged and enlarged. Both the right and left lobes were swollen and rounded like passive congestion. On freeing the adhesions between the duodenum and the liver a small tear was made in the duodenum from which a small amount of yellow gelatinous material escaped. This opening was sutured and the exploration continued. No lesion was found in the stomach or duodenum. There was no evidence of malignancy. The liver was abnormally large. Its condition and the overlying edema seemed to be due to passive congestion. The incision was closed without further exploration.

FURTHER DISCUSSION

DR. CABOT: "Possible perforation of the stomach." Can that exist with such films as these, Dr. Holmes?

DR. HOLMES: I think not. In most perforations there is some free gas in the belly, and we should get gas between the liver and the diaphragm.

DR. CABOT: We know she has aortic regurgitation. We have to find what kind, rheumatic or syphilitic. On the whole I am going to say rheumatic. She had had an attack of rheumatism. That is much more common than the syphilitic type. We have no good evidence of syphilis anywhere. The enlarged left auricle makes it quite possible that there is a mitral lesion too, even though the clinical evidence is not put into this record. It cannot amount to much if we consider the jump of the blood pressure from 20 to 160, which is hardly consistent with much mitral lesion, because there is so little blood coming through a stenosed mitral valve that the left ventricle could not contract violently enough to produce such a systolic pressure. If there is any mitral lesion it should be slight, hardly to be considered. There will be hypertrophy and dilatation of the heart. So far as the x-ray goes it looks as though it were the left ventricle and the left auricle. We have no electrocardiogram. The kidneys will show nothing, the lungs nothing. The liver may be cirrhotic, but they did not find it so at operation. They certainly had a good chance to look. I do not know what else there is. I do not think there is anythng else except congestion of the liver. Perhaps we shall never know the cause of her pain.

A STUDENT: In an early alcoholic cirrhosis don't you get some suggestion of tenderness in the liver?

DR. CABOT: I would not deny the possibility of that. I never saw an early one. This one is late I suppose, because if it is that the cause for it has been going on a long while.

A STUDENT: Would operation rule out the possibility of obstruction of the common duct?

DR. CABOT: I should say so. I cannot imagine it was not done with such thoroughness as to rule that out.

CLINICAL DIAGNOSIS

Rheumatic heart disease with congestive failure.

DR. RICHARD C. CABOT'S DIAGNOSES

Rheumatic heart disease, aortic stenosis and regurgitation.
Hypertrophy and dilatation of the heart.
Chronic passive congestion of the liver.

ANATOMIC DIAGNOSES

1. *Primary diseases.*

Aortitis, probably syphilitic.
Acute and chronic endocarditis, rheumatic and syphilitic.

2. *Secondary or terminal lesions.*

Chronic passive congestion of the liver.
Central necrosis of the liver.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The autopsy brings some surprises. The one outstanding anatomic lesion was the most extensive acute luetic aortitis that I have ever seen. Histologically the process is typical of syphilis and does not suggest rheumatic aortitis. The heart was slightly enlarged, weighing 425 grams. The aortic valve showed very slight fibrosis and slight separation of the cusps; there may possibly have been slight regurgitation. The mitral valve showed a few minute verrucose vegetations but no chronic lesion. There was no marked degree of deformity of any valve. The orifice of the right coronary artery was definitely narrowed.

Many adhesions around the bed of the gall bladder and the duodenum were still there. I do not believe the surgeon succeeded in freeing very many of them. There was marked chronic passive congestion of the liver and a very slight degree of central necrosis. I have no proof as to what caused her pain, but the findings in the aorta make me think of gastric crises more than anything else. It is not entirely impossible that those attacks of "cholecystitis" and "appendicitis" may be explained on the same basis.

DR. CABOT: There is no other lesion of syphilis besides that, then?

DR. MALLORY: No, and I cannot demonstrate spirochetes in the aorta. But the Levaditi stain is not always reliable, and it is not at all impossible that we may find them yet. I have never seen such extensive aortitis due to rheumatic fever. It can cause aortitis, but it does not produce such extensive destruction of tissue, and the cellular infiltration is different in character and distribution. The mycotic type of aortitis can also be readily distinguished, and I feel I can rule it out in this case.

DR. HOLMES: Is it confined to the thoracic aorta?

DR. MALLORY: Largely, though about four centimeters of the upper abdominal aorta was also involved.

DR. HOLMES: Was the aorta dilated?

DR. MALLORY: Somewhat diffusely dilated. The process began just above the valve, which was slightly involved.

A STUDENT: Were there any Aschoff bodies?

DR. MALLORY: I thought I saw a questionable one around one artery in the heart. There is no suggestion of them in the aorta. They are always unsatisfactory to look for in the average routine sections. If we examined fifty sections of every heart instead of one or two we should find many more of them.

DR. HOLMES: Was there any evidence of tuberculosis in the lung?

DR. MALLORY: There is none recorded, but I think there must have been a small apical lesion. They are so nearly universal that we sometimes neglect to mention them.

A STUDENT: Isn't it rather unusual for neurologic syphilis to go along with vascular syphilis?

DR. MALLORY: Yes, I think that is very true, but the two are occasionally found together.

A STUDENT: How do you explain the Corrigan pulse and the blood pressure of 160/20?

DR. CABOT: Likewise the dyspnea?

DR. MALLORY: There is no question but that she must have had some leakage through the aortic valve in spite of the apparently slight anatomic lesion.

A STUDENT: Then you can discount the statements about that pistol shot?

DR. MALLORY: On the contrary, I am giving them more credit than my own anatomic findings.

A STUDENT: What do you think was the cause of death?

DR. MALLORY: I do not know; she had heart disease without question, and an element of postoperative shock may well have been present.

A STUDENT: Can you do a Wassermann postmortem?

DR. MALLORY: Not with any reliability.

A STUDENT: Does syphilis explain the central necrosis?

DR. MALLORY: No. Acute endocarditis might, however.

DR. CABOT: You did not hear any account of why they did not have a Wassermann, did you?

DR. MALLORY: No.

A STUDENT: Could she have had any infarctions at all?

DR. MALLORY: We found none.

A STUDENT: What caused the congestion of the liver?

DR. MALLORY: Heart failure. I do not mean to say she did not have heart disease. I think it was probably both rheumatic and syphilitic at the same time. I am, however, much surprised that the anatomic evidence of it is so slight.

DR. HOLMES: There is one other interesting thing regarding this case. You find one of the causes of deformities of the duodenum as given in the textbooks is adhesions,—very extensive adhesions around the duodenum. I rather doubt that statement, because adhesions are not likely to cause deformities.

DR. CABOT: I think Dr. Mallory would agree that one cannot ask a pathologist to leave out of account clinical facts in making a decision for

or against aortic regurgitation. We wish we could prove it post mortem, but we do not find evidence that it was not there. And I suppose it is very much the most definite lesion that is present in this case.

Certainly the case does not make one eager to favor operations for adhesions. I think the more experience you get in that method the less you want to favor operations for adhesions, which so far as I know is all the diagnosis they had. We cannot possibly blink the fact that this patient's death was caused by operation.

A STUDENT: Couldn't the stiffness of the vessels in this extensive aortitis have caused a marked difference between the systolic and the diastolic pressure?

DR. CABOT: That is, syphilis of the aorta?

A STUDENT: Yes. Couldn't you account for the pulse pressure by the lack of elasticity of the vessels?

DR. CABOT: I do not see how that could cause the systolic pressure to go up to 160. You must have some elasticity to have that. I have never seen a pressure like this in arteriosclerosis.

A STUDENT: Could the pain be associated with displacement of the esophagus?

DR. CABOT: I do not believe so. What do you say?

DR. HOLMES: I think not. I was wondering, Dr. Mallory, if that could be associated with coronary disease?

DR. MALLORY: Yes, I think that is quite possible.

DR. CABOT: We do not get angina as low as the epigastrium. In view of this coronary finding that has to be considered too.

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VACCINATION

As the time approaches for the consideration of desirable public health legislation, the question of the wisdom of presenting bills to improve the vaccination law will come up for decision.

The arguments founded on existing conditions are growing stronger because the United States Public Health Service has stated that during ten months of the current year about 40,700 cases of smallpox have been reported in this country, whereas during the corresponding period of last year about 28,000 cases were recorded. Even the larger figures probably do not make the situation sufficiently impressive because it is well known that all cases are not reported.

The United States and British India report a larger incidence of smallpox than any other country. Records show that since 1925 there has been a gradually increasing number of cases, the figures for 1926 being 33,732, those for 1927 show an increase of 3,760; 1928 had 39,396 and 1929 had 42,282 cases. The cases to be reported in the remainder of 1930 according to precedent will probably make the total exceed the figures for 1929. The rates of incidence in smallpox show that the 1926 figures are .288 per 1000 of the population and the 1929 figures are .348.

Even available statistics may be very misleading for in the districts from which reports are not made regularly, or in some cases not made at all, are the districts where smallpox is most prevalent.

It is in these sections where the greatest carelessness with respect to prevention prevails. It may be that there are twice as many cases of the disease as are reported. It is not improbable that taking into account the average life expectancy, it might be that one person in twenty-five is likely to have an attack of smallpox during his lifetime.

Although the mortality from smallpox is less than one per cent. at the present time, history has shown that different epidemics have varying mortality rates and the development or importation of some virulent strain might bring about a mortality of thirty per cent.

Our protection consists in vaccination and the records show that at continental and insular stations and at foreign ports there were 203,388 persons vaccinated last year. This large group acts as a protective barrier against the disease and added to this are the persons vaccinated because of belief in the efficacy of the procedure and those who are vaccinated under State laws.

The protective power of vaccination is diminished in proportion to the number of unvaccinated people in a community. In Massachusetts there are two weak places in the chain, first in the number of pupils in private schools where vaccination is not required and second among the cases certified by physicians as unsuitable for vaccination.

The greatest advance would follow vaccination of all infants and revaccination at regular intervals thereafter.

A LAYMAN'S INTEREST IN MEDICINE

MR. EARLE P. CHARLTON who, by reason of unusual business ability and industry amassed a large fortune, has shown his appreciation of the importance of medicine and the service rendered by physicians by incorporating in his will an endowment of $500,000 for the Truesdale Hospital in Fall River in addition to a direct bequest to this hospital to be used in cancellation of debts accrued previous to January 1, 1926.

Tufts College will receive one-third of about $1,000,000 for research work in the medical department.

Other medical institutions mentioned are the Union Hospital of Fall River and the Johns Hopkins Hospital.

This is an illustration of the educational in-

fluence of hospitals and practitioners of medicine in attracting the attention of successful business men to the wisdom of supplying adequate support for institutions devoted to the alleviation of human suffering.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

FORSBECK, FILIP C. B.S., M.S., M.D. Rush Medical College 1925. Formerly Epidemiologist, Division of Communicable Diseases, State Department of Health. Assistant, Rockefeller Institute for Medical Research. Address: Rockefeller Institute for Medical Research, 66th Street and Avenue A., New York, New York. Associated with him is:

LUTHER, ELIOT H. A.B., M.D. Harvard Medical School 1921. Formerly Epidemiologist, Division of Communicable Diseases, State Department of Health. Address: Saranac Lake, New York. Their subject is: "Anterior Poliomyelitis in Massachusetts 1907-1929." Page 1115.

PARVEY, BENJAMIN. M.D. Baltimore Medical School, University of Maryland 1907 and University of Vienna and Budapest, 1928-1929. Member of Staff, Boston Dispensary. His subject is: "Early Diagnosis of Pregnancy: Experience with the Zondek-Aschheim Test." Page 1121. Address: 311 Commonwealth Avenue, Boston.

WILLIAMS, LINSLY R. A.B., A.M., M.D. Columbia 1899. Director of New York Academy of Medicine and President of the New York Tuberculosis and Health Association. Address: 5th Avenue and 103rd Street, New York City. Associated with him is:

HILL, ALICE M. A.B., Statistical Field Service, National Tuberculosis Association, New York City. Address: 370 Seventh Avenue, New York City. Their subject is: "The Patient's Reaction to a Diagnosis of Tuberculosis." Page 1129.

PHANEUF, LOUIS E. Phm.D., Ph.C., M.D. Tufts College Medical School 1913. F.A.C.S. Professor of Gynecology, Tufts College Medical School. Surgeon-in-Chief, Gynecological and Obstetrical Department, Carney Hospital. His subject is: "Two New Clamps for Abdominal Panhysterectomy." Page 1131. Address: 270 Commonwealth Avenue, Boston.

QUINCY, JOSIAH E. B.S., M.D. Northwestern University Medical School 1922. Clinical Assistant, Massachusetts Eye and Ear Infirmary. Junior Assistant Surgeon, Children's Hospital. Assistant Laryngologist, Faulkner Hospital. His subject is: "A Useful Tongue Depressor." Page 1132. Address: 270 Commonwealth Avenue, Boston.

ALLEN, FREDERICK M. A.B., M.D. University of California 1907. Formerly Research Fellow, Harvard Medical School and Associate in Medicine, Rockefeller Institute, New York City. Now Director of Physiatric Institute, Morristown, New Jersey. His subject is: "Methods and Results of Diabetic Treatment." Page 1133. Address: The Physiatric Institute, Morristown, New Jersey.

ROBINSON, RICHARD W. A.B., M.D. Yale University, New Haven, Conn. 1920. His subject is: "Pyuria in Children." Page 1139. Address: Laconia, New Hampshire.

OTIS, EDWARD O. A.B., Sc.D. (Hon.) Tufts College 1924 and New Hampshire University 1924. M.D. Harvard 1877. Professor of Medicine (Emeritus) Tufts College Medical School. Honorary Director, National Tuberculosis Association. Honorary President, Massachusetts Tuberculosis League. His subject is: "The Prevention of Disease, Some Applications of Preventive Medicine." Page 1143. Address: 81 Front Street, Exeter, New Hampshire.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY

A. K. Paine, M.D., *Chairman* — Fred. J. Lynch, M.D., *Secretary*

M. F. Eades, M.D., *Clerk*

What special treatment should pregnancy complicating tuberculosis receive?

In answering this question one must take into account the teachings of two different schools with ideas entirely opposed. As a general principle, the more conservative views, advocated by the French, seem to be gaining ground, many authorities declining an intervention and others holding that it is indicated only in exceptional cases.

The German teaching has been that tuberculosis complicating pregnancy undergoes exacerbation and that, in the interest of the mother, abortion should be performed when tuberculosis is detected during the first five months. When recognized later, intervention is not indicated since the induction of premature labor gives rise to results as serious as delivery at term.

In 1926, Bridgman and Norwood[1] studied 34 cases in Williams' service and showed in a follow-up of one year or longer that more deaths had occurred in the cases aborted than in those which had been allowed to go on with the pregnancy.

In 1925, Forssner[2] of Stockholm, aided by several specialists, showed that pregnancy exerted little, if any, influence on tuberculosis. He compared the results of 341 women who had been delivered with those of 396 other women who had been treated in the tuberculosis dispensary

but who had not been pregnant. At the end of two years, it was found that the mortality was essentially identical in each group. *Forssner believes that intervention is never indicated, and when practiced, probably does more harm than good.*

Nursing should not be allowed in the interests of both mother and child. Women who have done well during pregnancy grow rapidly worse after delivery, if they nurse their children.

Tuberculosis of the mother, as a rule, does not affect the child and it is not unusual for it to be very well developed. On the other hand, the disease is occasionally transmitted in utero so that the child is born with congenital tuberculosis. According to Williams[3], Whitman and Green in 1922, collected 38 cases with characteristic, histological findings, as well as 21 others in which bacilli were found in the fetus and placenta without histological lesions.

Couvelaire[4], on the other hand, believes that placental transmission occurs relatively frequently and bases his belief upon the existence of the so-called "ultravirus" of Calmette[5]. The latter holds that tuberculosis may be transmitted not only by the actual bacillus, but also by a virus which can pass through a Chamberland filter and from which bacilli develop later. It appears, however (Williams[3]), that in the vast majority of cases the disease is not transmitted directly through the placenta to the fetus, but that the latter is born with a lowered resistance rather than with tuberculosis itself. Hence, it follows that such children should be brought up under the best hygienic surroundings and should not be put to their mothers' breasts.

Lanz[6], divides the tuberculous lesions of the placenta into four forms as follows: (1), tubercles attached to the periphery of the chorionic villi, but projecting in great part into the intervillous spaces; (2), tubercles in the stroma of the villi, which have penetrated the villous epithelium; (3), tubercular changes in the chorionic membrane and (4), caseous tubercular deposits in the decidua basalis. Of these, one and four are the most frequent while two, which is the lesion most concerned in the production of congenital tuberculosis, has only occasionally been observed.

DeLee[7], states that a great deal depends on the stage of the infection when gestation begins. *"A woman with healed tuberculosis may safely become pregnant."* The author summarizes the treatment as follows: Tuberculous women may not marry because thereby they aggravate their own disease, infect their husbands and propagate tuberculous children. During pregnancy the medical treatment of tuberculosis should be carried out. On the interruption of pregnancy opinions are divided. Three positions are held: 1, that pregnancy should always be interrupted as a curative measure; 2, that it be never interrupted; and 3, that the cases be individually treated and the gestation terminated only when certain symptoms indicate it. DeLee is more inclined to the active treatment.

Trembley, of Saranac Lake, quoted by DeLee[7], induces abortion in the early months in all cases, and says that the mortality and percentage of relapses are practically nil when abortion is induced before the end of the third month. Morris, from a study of 1000 cases, came to the same conclusions.

If the process is very chronic, especially if the patient comes in the second half of pregnancy it is justifiable to wait and watch closely. Urgent symptoms of cardiac nature, persistent hemoptysis and dyspnea may require the termination of gestation. Complicating nephritis, heart disease and contracted pelvis will give earlier indications for interference. Tuberculosis with hyperemesis and laryngeal tuberculosis in the early months, are positive indications for interruption. Finally if one kidney is tuberculous it may be removed, if both, abortion is indicated.

DeLee[7], feels that abortion, when indicated, should be performed in two states as follows: By the administration of morphine and scopolamine, by the opening of the ovum and by packing the uterus with gauze. On the following day the products are removed, if they have not come away spontaneously. Premature labor is best induced by rupturing the membranes, metreurysis or vaginal Cesarean section. Local anesthesia with novocaine should be employed whenever possible. General anesthesia is contraindicated as it spreads the process in the lungs. During labor, watchful expectancy should be the rule, being ready for emergencies, the main attention being given to the heart. The parturient is best delivered with forceps when the head appears on the perineum.

The hemorrhage of the third stage should be limited. DeLee holds that ethylene is the best form of anesthesia when morphine, scopolamine, or local cannot be employed. During the puerperium, caffeine should be prescribed for the heart, the mother should not nurse the child which should be removed from the environment.

In 1921 a separate clinic for the treatment of tuberculous women in the pregnant state was established at the Maternité Baudelocque. The tuberculous patients are isolated in the Tarnier Pavilion, a building entirely separated from the main clinic, during pregnancy, labor and the puerperium. The new-born are immediately isolated and are not allowed to nurse. The close collaboration between Couvelaire[8], Leon Bernard and Rist, has shown the excellent results obtained by purely conservative treatment.

REFERENCES

1 Bridgman and Norwood: Pulmonary Tuberculosis and Pregnancy. Johns Hopkins Hosp. Bull., Balt., 1926, 38: 83-111.
2 Forssner: Les relations entre l'état de gestation et la tuberculose. Acta. Gyn. Second, 1925, 3: 256-285.
3 Williams: Obstetrics. 6th edition. D. Appleton & Co., New York-London. 1930: 589-591.

4 Couvelaire: Le nouveau-né issue de mère tuberculeuse. Gynéc. et obst., 1927, 15: 1-9.
5 Calmette: Valtis et Lacomme. Transmission intrautérine du virus tuberculeux de la mère à l'enfant. Presse méd., Par., 1926: 1409.
6 Lanz: Ueber einen Fall von Tuberculose der Plazenta u. der Eihäute. Arch. f. Gynaek., Berl., 1915, 104: 238-258.
7 DeLee: Principles and Practice of Obstetrics, 5th edition, W. B. Saunders Company, Philadelphia-London, 1929: 522-524.
8 Couvelaire: La nouvelle Maternité Baudelocque. Masson & Cie., Par., 1930: 18-20.

Questions of a similar nature to the above will be discussed in the *Journal* each week. They may be addressed to the Clerk of the Committee in care of the *Journal* and will be answered by members of the Committee of the Section of Obstetrics and Gynecology.

THE COMMITTEE TO WHOM WAS REFERRED "THE SUBJECT OF PREPARING NOTICES OF DECEASED FELLOWS" HAVE ATTENDED TO THAT DUTY AND BEG LEAVE TO SUBMIT THE FOLLOWING REPORT*

It seems that no provision is made in the By-Laws or Rules of the Massachusetts Medical Society for preparing and publishing any obituary notices of its members, and from an examination of its printed Records it does not appear that any action was ever taken in the matter, and the notices of this character that have been published, have been the volunteer labor of the individual appointed to give the Annual Address, in conjunction with the personal friends of deceased members.

In the year 1843 notices of this kind first appeared appended to the Annual Discourse, and since that time, in ten of the twenty-five published reports, no such notices are found. And though there have been on an average annually from 25 to 30 deaths only about one-third of these have been particularly noticed. While some of the notices have been very complete and discriminating, other deceased members equally deserving and perhaps more distinguished receive no notice at all. There has evidently been no system or uniformity in the matter.

It will be seen at once how this happens, as the Orator is not charged with this duty and has enough else to do, and no friends of the person deceased always appearing to make such contributions.

There can be, we think, but one opinion, upon this subject—that is, in an Association as large, as respectable and composed wholly of educated men as the Massachusetts Medical Society is, there should be some provision made whereby all its deceased members shall be properly noticed at its anniversary meetings or in its published reports. No matter how humble or obscure the member may be, a few lines should be devoted to his history, character and death. Such is the practice now with most of our large literary Institutions as well as in Associations kindred to this Society.

The question is how can this be best done; can it be by any officer already chosen—say by the Orator or by one of the Secretaries or shall an individual or a Committee be appointed for this purpose?

Your Committee, after much reflection, would recommend that a permanent office of this character be created, and that some individual shall be chosen by ballot at the Annual Meetings of the Society for this place. And that the Secretaries of the several District Societies be requested to communicate to this Officer, from time to time, upon the decease of members in their respective Societies, such facts as may be desirable to incorporate into obituary notices.

N. Allen, *Chairman of Committee.*

*Found in the archives of the Massachusetts Medical Society and submitted for publication by the Secretary, Dr. Walter L. Burrage.
Dr. Allen joined the Society in 1842 and died in 1889.

THE BOSTON MEDICAL LIBRARY

VALENTINE MOTT, M.D.
1785-1865

Conspicuous among the makers of surgical history in America is the name of Valentine Mott. His forbears were English, the first one to come to America being Adam Mott, who settled on Long Island about the middle of the seventeenth century. He was a Quaker, a disciple of George Fox, and from such ancestry it is not surprising that strong religious bias should have been exhibited by his descendants. Valentine Mott's father, Avery Mott, was a physician who practised for some time in New York city and was a pupil of the elder Dr. Bard, a famous practitioner and one of the earliest to demonstrate anatomy on the human cadaver in this country. Valentine Mott was born in Glen Cove, Long Island in 1785, just after the close of the Revolutionary War. Very little is known of his boyhood other than that he acquired his classical education in a private Seminary at Newtown. One can only conjecture what sort of a character he must have possessed at this time from his behavior in later life. When he was about nineteen he began the study of medicine with a preceptor, Dr. Valentine Seaman, who was also a kinsman. During this period he attended two full courses of lectures in Columbia University and was granted his medical degree in 1807. It was customary then to offer a thesis as part of the qualification for a degree and he chose for his subject "An Enquiry into the Chemical and Medicinal properties of Statice Lunonium" (Marsh Rosemary). Seaman was a pupil at the University of Pennsylvania under Rush, Shippen and Kuhne, was a surgeon to the New York Hospital and one of those instrumental in the introduction of vaccination into America. As a medical student young Mott was industrious, devoting much time to Anatomy and Surgery and

was graduated with high honor. Shortly after he obtained his degree he departed for Great Britain where he spent the next two and one-half years, i.e., until 1809, when he returned to New York. A part of this time he studied in London under Sir Astley Cooper, Abernethy, Cline and Charles Bell. From here he went to Edinburgh, where he came under the influence of a brilliant group of Scottish clinicians active in the University at that time, viz.; Gregory, Black, Munro, Duncan, Home, Hope and Thomson. To these must be added the names of John Bell and Dugald Stuart, the latter, a teacher of philosophy, made a lasting impression upon Mott and to him he frequently refers in the most enthusiastic terms. In Edinburgh at that time were also several literary lights who served to render the atmosphere of the city extraordinarily stimulating.

The winter following his return to New York he delivered a course of private lectures on Surgery and was shortly thereafter appointed to the professorship of Surgery at his Alma Mater, Columbia College. During all the vicissitudes through which the medical school passed he still held this position and was associated with a brilliant group of teachers who withdrew in a body from the College of Physicians, when the trustees became tyrannical, and formed a new school in connection with Rutgers College. This was in 1826. For five years this school continued to grant degrees in medicine but was finally compelled to close its doors because of a technical illegality in the conference of degrees. He then went back to the College of Physicians and Surgeons as Professor of Operative Surgery and Pathological Anatomy. On account of ill health he resigned in 1835, spending some time in foreign travel. In 1840 he accepted the position of Professor of Surgery and President of the Medical Faculty of New York University. The school prospered, large numbers of students from all over America and some foreign countries seeking admission. In 1850 he resigned to make a second European visit. In the fall of that year, upon his return, he resumed his connection with the College of Physicians, this time as Professor of Operative Surgery and Surgical Anatomy. At his death in 1865 he was Emeritus Professor of Surgery at the College of Physicians and Surgeons.

These various fluctuations in the fortunes of the medical schools were vexatious in the extreme but were in no way his fault. His colleagues during the period of his activity in New York practice were Hosack, Delafield, Francis, Macneven, Wright Post, Mitchell, Godeman and Griscom. His reputation as a surgeon had spread beyond the confines of his own country because of the brilliancy of his operative technique, for he was not much given to writing. In consequence of this fact he was enthusiastically received wherever he went. He first sought out Sir Astley Cooper when he reached London, for it was from him he received his first postgraduate instruction. From there he went to Scotland, France, Belgium, Germany, France, Ireland, Italy, Greece, Egypt, Turkey and Asia Minor. When in Constantinople he operated upon the Sultan. He was much criticised at home for sacrificing a Cock to Aesculapius at Epidaurus, the birthplace of Aesculapius. The sacrifice was part of a clinical lecture, probably the first ever given in that country, at which he tied both the carotid arteries of the victim. During his trip he met Larrey, Napoleon's surgeon, von Graefe, Dieffenbach, Velpeau and many others. It was at this time that he became interested in orthopaedic surgery due to the fact that Stromeyer had just introduced the operation of tenotomy. He was fired with a desire to found a hospital for the care of orthopaedic cases at Bloomingdale but was persuaded to give up the idea because his friends feared such a move would not be popular with the profession. Some of his notable surgical triumphs were the ligation of the innominate artery for aneurism, resection of one-half the lower jaw for a sarcoma, amputation at the hip joint, one of the first to have been performed in this country, excision of the clavicle, spina bifida, lithotrity and the ligation of most of the large arteries of the body for various causes. In all he performed this operation of ligation 58 times. He was the first to tie the iliac artery for aneurism and resect the lower jaw for necrosis. He ligated the common carotid forty-six times; cut for stone 165 times and amputated nearly 1000 limbs. Sir Astley Cooper said of him, "he has performed more of the great operations than any man living, or that ever did live". He introduced into American medical teaching the principle of bedside instruction in surgery.

His public contributions to surgical literature comprise about twenty-eight papers. His original surgical operations number seven. His abilities were widely recognized abroad. Edinburgh granted him a degree in medicine; he was made a Fellow of the Imperial Academy of Paris, of the Chirurgical Society of Paris and Fellow of the Medical and Chirurgical Societies of London and Brussels. King and Queens College of Physicians of Ireland filled a vacancy, caused by death, by electing Dr. Mott as an Honorary Fellow. This was all the more complimentary in view of the fact that this Society had elected but 20 members in the preceding two hundred years. In recognition of his skill in removing a tumor of the scalp from the Sultan of Turkey he was knighted. His interest in anatomy, particularly regional surgical anatomy, was an engrossing one all his life. Pathological anatomy also claimed his attention and he had a private museum of specimens of this sort which was more extensive than many connected with hospitals and teaching institutions. His habits of

neatness in personal appearance and punctuality to all appointments of whatever sort were proverbial. Though keenly interested in public affairs and well informed upon them, he is said rarely to have cast a vote at an election. It is difficult to understand how this could be true in a character otherwise so exemplary. Under all circumstances he was the courteous gentleman of the "old school". He had a large family, two of the sons going into medicine. His practice was extensive and for the time, a very lucrative one, particularly in the later years when his reputation was such that large fees were readily collected. His estate was valued at about a million dollars, which for a professional man was quite as unusual then as now. He did not confine himself to surgery, in fact at one time he had one of the large obstetric practices in the city. His life and accomplishments furnish a striking illustration of what the persistent application of average intellectual power may bring about when directed to the attainment of a definite end.

MISCELLANY

1930 SEAL SALE

Seventy-eight million Christmas Seals sent out by the Affiliated Organizations of the Massachusetts Tuberculosis League have been placed on sale. This number exceeds the number of Seals placed on sale in any previous years by five millions. The National Tuberculosis Association, which sponsors the Seal Sale throughout the United States, states that the demand for Seals this year has exceeded that of any of the preceding years.

The 1930 Seal bears the picture of Santa Claus and the double-barred cross, copyrighted emblem of the tuberculosis campaign.

Confusion sometimes occurs over the Red Cross Membership Campaign which begins on Armistice Day and continues until Thanksgiving Day and the Tuberculosis Seal Sale which is carried on throughout December. These two National Organizations are distinctly different. The Tuberculosis Associations confine themselves exclusively to the campaign against tuberculosis by the employment of nurses, by conducting summer health camps for undernourished children and by a campaign of health education for the prevention of the disease.

THE RICHNESS OF MILK IS NO GUARANTEE OF ITS SAFETY

Although health authorities have for years been stressing the advisability of pasteuring or boiling for the purpose of making milk safe for consumption, it is evident that most people judge the quality of milk by the amount of cream which is apparent in the container. While cream is of importance as an evidence of butter fat, it is without value as an indicator of the purity of the product.

Those outside of city boundaries are not deeply concerned with the quality of milk from the standpoint of bacterial invasion, although very much has been done in an effort to educate the people with reference to the importance of milk free from tuberculosis and streptococci.

Because these things are not understood and cannot be seen by those who use the milk, great difficulty has been encountered in securing endorsement of the recommendations of health authorities.

It has been demonstrated repeatedly that a high-grade product can be produced at very little greater expense than under ordinary methods. When the time shall have come that the importance of wholesome milk is recognized, there will be a great diminution in certain types of illness.

A CRITICISM OF METHODS INCIDENT TO THE ENROLLMENT OF STUDENTS IN MEDICAL SCHOOLS

At the opening of a campaign to establish a college of medicine at Long Island University, Dean George R. Hardie, Dr. Rypins, Secretary of the New York State Board of Medical Examiners, and Dr. S. J. Kopetzky, Editor of the *Medical Week*, criticised the methods of passing on applicants for admission to medical schools.

The objections expressed were based on beliefs that the matriculation quota of some of the established medical colleges is determined on the "basis of racial, religious and geographical distribution of applicants" and certain aristocratic tendencies in the professions of medicine, law and dentistry which are contrary to the principles of democracy.

The evident purpose of the proponents of this new medical school is to make it possible for large numbers of young people to enter upon the study of medicine.

FUND FOR THE GOOD SAMARITAN HOSPITAL REACHES A TOTAL OF $114,099

Gifts totaling $114,099 toward the half-million fund being sought to provide rheumatic fever research at the Good Samaritan Hospital were announced Tuesday, December 2, at a report luncheon of the women's committee, at Perkins Hall, Women's Educational and Industrial Union. Mrs. Henry B. Chapin, chairman, presided.

With 1,000,000 children in the United States suffering with weak or damaged hearts, the hospital is not content merely to help those who can be accommodated as patients, the speakers pointed out, but it feels it must strive to find a prevention and cure which will be of service to sufferers throughout the world for this unconquered malady. Research must be provided to throw more light on this little understood disease which is growing worse each year, particularly in Massachusetts.

Mrs. William Stanley Parker, a vice-chairman of the women's committee, at the luncheon held November 24 said that the recent report of Public Health Service stated that "of the 150,000 to 200,000 persons,

usually children or adolescents, who are attacked by rheumatic fever every year, no less than 40 per cent. develop diseases of the heart. If rheumatic fever could be prevented, we would at the same time prevent about 80 per cent. of the cases of chronic heart disease."

"The Good Samaritan, for ten years, has devoted its chief attention to providing the best possible care for women and children with rheumatic heart disease. It is doing a work not being done at the Children's Hospital in keeping patients over a prolonged period. The Good Samaritan relieves beds in the Children's Hospital, in the City Hospital, the Massachusetts General and other institutions for patients more acutely ill, and works in the closest harmony with these institutions, in no way duplicating their work," Mrs. Parker explained.

"But to continue indefinitely just ministering to a limited number of patients is not taking advantage of the exceptional opportunity the Good Samaritan has to help check this disease for sufferers everywhere. Yellow fever, malaria, tuberculosis, diphtheria and scarlet fever—think how terrifying these diseases were before medical science found ways to check their ravages!"

Dr. Levine said: "Heart disease today is the most common cause of invalidism and death in this country. It is also the greatest cause of economic loss through illness amongst our citizens. This problem is particularly devastating when it takes the form of rheumatic heart disease, for then the young and middle-aged are the victims."

"New England suffers especially in this regard," Dr. Levine added, as rheumatic heart disease is most prevalent in this part of the country. It seems to me that the time has come for more determined and concerted effort to be directed at these fundamental questions.

"The Good Samaritan Hospital has been a pioneer in so many important medical matters in the past that it deserves the most generous support in this new project of combating heart disease in the young. No other hospital is giving the long-time care to rheumatic fever patients which they receive there."

CORRESPONDENCE

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn St., Chicago, Ill.
November 29, 1930.

Managing Editor,
The *New England Journal of Medicine*,
165 Newbury St.
Boston, Mass.

In addition to the articles enumerated in our letter of October 24 the following have been accepted:

Abbott Laboratories
Gold Sodium Thiosulphate—Abbott

Eli Lilly & Co.
Erysipelas Antistreptococcic Serum—Lilly (Concentrated Globulin)

Medical Arts Laboratory, Inc.
Antirabic Vaccine, Semple Method

National Drug Co.
Antipneumococcic Serum, Type I
Ointment Scarlet Red Biebrich 8 Per Cent.
Typhoid-Paratyphoid A Vaccine

Parke, Davis & Co.
Ventriculin

Richards Pharmacal Co., Inc.
Richards Psyllium Seed

E. R. Squibb & Sons
Diphtheria Toxoid-Squibb, twenty 1 cc. ampule packages
Diphtheria Toxoid-Squibb, two 1 cc. ampule packages
Normal Horse Serum, one 50 cc. vial package
Ragweed Pollen Allergen Solution-Squibb (3 vial treatment package)
Timothy Pollen Allergen Solution-Squibb (3 vial treatment package)

W. A. PUCKNER, *Secretary*,
Council on Pharmacy and Chemistry.

RECENT DEATH

BRAINERD, WALTER SCOTT—In continuation of the notice of death of DR. WALTER SCOTT BRAINERD, information has been received that he practiced as a specialist in diseases of the eye, ear, nose and throat for which his preparation had been made by study in England, France, Germany and other countries.

He was born in Black River Falls, Wisconsin, in 1859, the son of Fredus and Maria Washburn Brainerd. His preliminary education was acquired in the elementary schools in China, Maine, and the China Academy. He entered the University of Vermont from which he graduated in 1883. His first field of practice was in Pemaquid, Maine, where he remained for about twenty-two years. During this period he was active in the civic life of the community, having served as selectman and town clerk for several years. In 1884 he attended the New York Post-Graduate Medical School paying particular attention to pathology, physical diagnosis and clinical medicine. In 1905 he studied at the Central London Throat and Ear Hospital where he was clinical assistant on the Hospital Staff and served on several committees. He took an additional course in Berlin and Vienna in the treatment of diseases of the eye and at the London Royal Ophthalmic Hospital. He served on the Staff of the Gale Hospital in Haverhill for several years during his twenty-four years' residence in that city.

He was a member of the American Medical Association, Pentucket Medical Association and the Massachusetts Medical Society. He was a Mason

and a member of the First Church of Christ at Bradford.

He is survived by his wife, Mrs. Elizabeth W. (Ward) Brainerd; three daughters, Mrs. George B. Whitney, Haverhill, Miss Katherine E. Brainerd, Haverhill, and Mrs. Evert A. Bancker, Atlanta, Georgia; four sons, Arthur T. Brainerd, Chicago, Ill.; Newell F. Brainerd, Haverhill; Walter E. Brainerd, Des Moines, Iowa, and Carl E. Brainerd, Albany, New York; two sisters, Miss Estelle Brainerd and Miss Helen Brainerd both of China, Maine, and eight grandchildren.

NOTICES

THE JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE

INSTITUTE OF THE HISTORY OF MEDICINE

A series of lectures at the Institute of History of Medicine at the Johns Hopkins School of Medicine was inaugurated recently. The remaining lectures are as follows:

I. Sir D'Arcy Power, of London, England, Visiting Lecturer in the History of Medicine.

Lectures December 10 and 17, 5 P. M.

II. Dr. William H. Welch, Professor of the History of Medicine.

Lectures or conferences on selected topics, on Wednesdays, 5-6 P. M., during January, February and March, beginning January 7, 1931.

III. Dr. Fielding H. Garrison, Resident Lecturer in the History of Medicine.

A seminar course on Medical Literature and Bibliography, on Thursdays, 5-6 p. m., began October 16, 1930, and will end January 22, 1931.

Lectures on Phases of National Medicine:

1. Russian Medicine: The Old Régime.
2. Russian Medicine: The Soviet.
3. Spanish Medicine.
4. Scandinavian Medicine.
5. Italian Medicine.
6. Germanic Medicine.
7. Medicine in France.

These lectures will be given on Thursdays, 5-6 P. M., beginning March 26, 1931, and ending May 7, 1931.

IV. Dr. Stephen d'Irsay, Associate in the History of Medicine.

Dr. d'Irsay will give at least one seminar course for advanced students (History of Physiology), as well as lectures on the History of Universities, and a course on Ancient and Mediaeval Scientific Textbooks.

V. Dr. John Rathbone Oliver, Associate in the History of Medicine.

Dr. Oliver will give an introductory course to the History of Medicine, on Tuesdays and Fridays, 3-4 P. M., during the second, third and fourth quarters of the academic year. The first lecture will be given on December 5, 1930.

These lectures are intended for students who have had no previous training in Medical History and who desire a brief summary of the entire field, from the medicine of earliest times down to medicine of the present day. These lectures will be illustrated so far as possible by lantern slides and by demonstrations of valuable medical works, photostats of manuscripts, etc.

The lectures will be given in the Lecture Room of the Institute of the History of Medicine, third floor of the William H. Welch Medical Library, 1900 East Monument Street, Baltimore, Md.

REMOVALS

Dr. Joseph H. McLaughlin announces the removal of his office from 482 Beacon Street to the Lister Building, 475 Commonwealth Avenue, Boston, Massachusetts.

Dr. Samuel Edelstein wishes to announce the removal of his office from 10 Newbury Street, Boston, to 471 Commonwealth Avenue, Boston.

RADIO MESSAGES

SPONSORED BY THE STATE DEPARTMENT OF PUBLIC HEALTH, COURTESY WBZA

Every Wednesday at 5:20 P. M.

December 10—"So-Called Chronic Appendicitis", Dr. Charles C. Lund.

December 17—"Accidents", Dr. Louis G. Howard.

December 24—"Some Diseases We Don't Talk About—And Why Not?" Dr. N. A. Nelson.

December 31—"The Hospital and the Public Health", Dr. Henry M. Pollock.

A "Health Forum" directed by the State Department of Public Health, courtesy WEEI, began October 2, 1930 and will continue Fridays at 4:50 P. M. Queries and Answers will be broadcast.

Questions on health and prevention of disease may be sent to "Forum", State Department of Public Health, State House, Boston, Mass.

REPORT AND NOTICES OF MEETINGS

PLYMOUTH DISTRICT MEDICAL SOCIETY

The November meeting of the Plymouth District Society was held in Grand Army Hall, Abington, Mass., November 20, 1930. The President of the Society, Dr. Frederick W. Murdock, was in the chair. The minutes of the preceding meeting were omitted. President Murdock appointed a committee to draw up resolutions on the late Dr. C. E. Lovell, consisting of Drs. Frame, Beaulieu, and Hanley, and one on the late Dr. Aurele Roy, consisting of Drs. Lemay, Champ, and Boucher. Dr. Thomas H. McCarthy, former president of the Society, then took the floor and stated that for the past two years the Legislative Committee

of the Plymouth District Society had invited the different Representatives and Senators in the Plymouth District to a meeting and dinner. The object was to explain to them the various medical and health bills in which the Massachusetts Medical Society was interested. He stated that the Plymouth method was highly commended by the Council of the Society, so much so, in fact, that a committee was appointed to use its influence to bring pressure upon all the different county societies of the Massachusetts Medical Society to conduct similar meetings so that all of the different Representatives and Senators would understand just what these bills were for. He stated also that our Society has been highly commended by the Council of the parent body, because we have tried in every way possible to advance the high ideals of our profession. They call it the Plymouth method because it originated in our Society. He then moved that a special meeting be held, similar to those of the past two years, and that the same methods be adopted at this meeting. Dr. John J. McNamara, Vice-President of the Plymouth District Society, seconded the motion which was carried.

Dr. Murdock, President of the Society, then introduced Dr. Soma Weiss, of the Harvard Medical School and the Boston City Hospital, who discussed "Present Concepts of Circulatory Failure."

Dr. Weiss emphasized that in circulatory failure there is a widespread disturbance of normal functions throughout the body, and that therefore the patient with heart disease must be studied as a whole. Narrow specialization in cardiovascular disease is not only unfair to the patient but also a handicap to the progress of knowledge of this condition. While electrocardiographic study of heart function has made fundamental contributions to the understanding of cardiac disease; once this understanding is attained, the electrocardiograph is not the most important instrument which may be used in general medical practice to evaluate the state of the circulation. In fact, indiscriminate use of the electrocardiograph is to be deplored. The shortcomings of this instrument are often best appreciated by those who are most familiar with its technique.

Dr. Weiss discussed the contributions which have been made to the understanding of cardiac disease during recent years through the use of other instruments than the electrocardiograph. These studies make it clear that progressive failure of the circulation cannot be explained on a basis of a uniform pattern as was believed in the past. There are several types of circulatory failure. Dr. Weiss gave examples of these types and stressed the importance to therapy of distinguishing between them.

A painstaking clinical analysis of the patient's condition is always more important than the use of complicated instruments; nevertheless, the significance of certain measurements should be pointed out. Dr. Weiss demonstrated by charts and lantern slides the various methods which may be used to measure the arterial and venous blood pressure, the vital capacity, cardiac output and velocity of blood flow. He discussed the values obtained in health and in disease, and demonstrated, so far as is possible, the interrelation of these measurements in various forms of circulatory disturbance. The combined measurements of the arterial and venous pressure and the vital capacity often give important indications as to the state of the circulation. The technique of measuring these aspects of the circulation is simple enough so that such measurements may be used in daily practice. Dr. Weiss demonstrated this technique in detail.

In closing, Dr. Weiss emphasized once more that the significance of instrumental studies of circulatory failure can never diminish the importance of bedside study of the patient. A history taken with intelligence, a physical examination performed with skill, and simple functional tests are the data on which the physician should base the answer to his fundamental questions: (a) is there organic heart disease present? (b) if there is organic heart disease, what is its etiology? (c) is the heart capable of performing its required daily work? A statistical analysis of such accumulated bedside observations certainly would lead to a better understanding of circulatory failure.

After Dr. Weiss finished his talk there was an open discussion, and his part of the program was discussed by Dr. William G. Walker, Dr. Thomas H. McCarthy, Dr. Frederick W. Murdock, Dr. Charles G. Miles, and Dr. H. A. Chase, who asked numerous questions in regard to his very interesting talk. Dr. Weiss gave one of the finest demonstrations of cardiac disturbances and their detection that has ever been given to the Plymouth District Society.

The meeting then adjourned. Among those present at the Society meeting were Dr. Edgar Hill, Medical Examiner from Plymouth, and Dr. Brown of Plymouth, who came into the meeting on crutches. It would seem that Dr. Brown had set an example for the different physicians in the district, because, if a man can take the trouble to come to a medical meeting on crutches, it does seem as though the other members of the Society should take advantage of this instructive and helpful address. There was a large attendance at this meeting. A fine luncheon was served by the Woman's Relief Corps of Abington after the meeting adjourned.

THE HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the amphitheatre of the Peter Bent Brigham Hospital at 8:15 on the evening of Tuesday, December 9.

PROGRAM

Presentation of Cases.

1. The Explanation of the Mysterious Emotional Increase of Heart Rate. Walter B. Cannon, M.D., Professor of Physiology, Harvard Medical School.

2. Some Aspects of Sulphur Metabolism. John M. Flynn, M.D., Peter Bent Brigham Hospital.

Physicians, nurses, and students are cordially invited to attend.

W. P. MURPHY, M.D., *Secretary.*

MASSACHUSETTS ITALIAN MEDICAL SOCIETY

The annual meeting of the Massachusetts Italian Medical Society will be held at the Hotel Kenmore, 496 Commonwealth Avenue, Boston (Telephone Kenmore 2770), on Monday evening, December 8, 1930. Dinner will be served at 6 o'clock sharp. The dinner will be followed by an address by A. L. Soresi, M.D., of New York City. The subject of the address will be:

"New Views on Pathogenesis, Diagnosis and Treatment of Ulcer and Cancer of Stomach, Cholelithiasis and Diseases of the Digestive Organs in General."

(Presentation of Specimens, X-Ray, Lantern Slides and, if possible, patients.)

Dr. Soresi is one of the pioneers in the above mentioned work. He has been selected from a long list of possibilities and we feel that the choice has been happily and properly made. We expect a good gathering to show our good feelings and courtesy to our invited guest.

The business meeting will follow after the address of our invited guest.

1. Reading of the minutes.
2. Report of Committees:
   a) Hospital Committee: Dr. Maraldi, Chairman
   b) Credential Committee: Dr. D'Errico, Chairman
   c) Public Health and Education Committee: Dr. Petrillo, Chairman
   d) Committee on Ethics: Dr. Barbarisi, Chairman
   e) Auditing Committee: Dr. F. Costanza, Chairman
   f) Nominating Committee: Dr. D'Errico, Chairman.
3. Amendment of By-Laws:
   On motion duly made and seconded let it be known that the body wishes that Art. 4, Section 1 of our By-Laws is to be changed to read as follows:
   "There shall be an annual meeting of the Society on the Second Monday of December of each year, and one meeting of the Society on the second Monday of each month, except in July and August."
4. Voting on above amendment.
5. Election of officers for 1931.

CARL F. MARALDI, M.D., *Secretary.*

THE WILLIAM HARVEY SOCIETY

FOUNDED BY THE STUDENTS OF TUFTS MEDICAL SCHOOL TO FURTHER INTEREST IN THE ART OF MEDICINE

Lecture at the Beth Israel Hospital

Friday, December 12, 1930, 8.00 P. M.

Speaker: Dr. Elliott P. Joslin, Clinical Professor in Medicine, Harvard Medical School.

Subject: "The Carbohydrates in the Body."

Chairman: Dr. David Rapport, Professor of Physiology, Tufts Medical School.

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held on Tuesday, December 16, 1930, at 12 noon, at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

The speaker will be William A. Hinton, M.D., Director of Research, Boston Dispensary, who will talk on "The Diagnosis of Primary Syphilis."

The usual luncheon will be served. All physicians are cordially invited to attend.

THE NEW ENGLAND PEDIATRIC SOCIETY

This Society will meet Friday, December 12, 1930.

PROGRAM

1. Clinical meeting conducted by the Children's Medical Service at the Massachusetts General Hospital—Dr. Harold Higgins and Associates, at 4:15 P. M.

2. Evening program at the Boston Medical Library at 8:15 P. M. "Nonpurulent Encephalitis in Children"—Dr. H. M. Zimmerman, Assistant Professor of Pathology, Yale University School of Medicine, and Dr. Herman Yannet, Assistant in Pediatrics, Yale University, School of Medicine.

Discussion by Dr. Tracy J. Putnam, Dr. Bronson Crothers and Dr. Charles F. McKhann.

3. Refreshments.

GERALD HOEFFEL, *Secretary.*

SOCIETY MEETINGS

CONGRESSES AND CONFERENCES

December 8—Massachusetts Italian Medical Society. Details elsewhere on this page.

December 9—The Harvard Medical Society. See page 1178.

December 10—Harvard Medical School. The George W. Gay Lecture on "Medical Ethics". Amphitheater C at 5 P. M. Dr. Robey.

December 11—Massachusetts General Hospital. See page 1110, issue of November 27.

December 12—The New England Pediatric Society. See notice above.

December 12—The William Harvey Society. Details elsewhere on this page.

December 15 and 17—Harvard University Medical School. Two Lectures on Recent Work on the Metabolism of the Eye. See page 1110, issue of November 27.

December 16—South End Medical Club. See notice above.

January 7—Cardiac Course. See page 854, issue of October 23.

March 23-27, 1931—Fifteenth annual clinical session of the American College of Physicians. Detailed notice appears on page 790, issue of April 17.

July 27-31, 1931—Third International Congress of Radiology, Paris. Complete notice appears on page 385, issue of August 21.

DISTRICT MEDICAL SOCIETIES

Bristol North District Medical Society

April 16, 1931—Taunton Woman's Club.
September 17, 1931.

Essex South District Medical Society

Wednesday, January 7, 1931—Beverly Hospital. Clinic 5 P. M. Dinner 7 P. M. Symposium on Duodenal Ulcer. Speakers: Dr. Franklin White, Medical Aspect. Dr. A. W. George, X-ray Aspect. Dr. P. P. Johnson, Surgical Aspect.

Wednesday, February 4, 1931—Council meeting, Boston.

Wednesday, February 11, 1931—Danvers State Hospital. Clinic at 5 P. M. Dinner 7 P. M. Speaker: Dr. Bryant E. Moulton, Assistant Director of the Judge Baker Foundation. Subject: "Juvenile Delinquency."

Wednesday, March 4, 1931—Lynn Hospital. Clinic at 5 P. M. Dinner at 7 P. M. Speaker: Dr. Arthur Allen, of Boston. Subject: "Neurovascular Surgery, including injections for varicose ulcer."

Thursday, May 7, 1931—Censors meet at Salem Hospital at 3:30 P. M.

Tuesday, May 12, 1931—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. Morris Fishbein, Editor of the A. M. A. Journal of Chicago. Subject: "History of Medical Cults." Ladies invited.

Franklin District Medical Society

January, 1931-May, 1931—The meetings are held on the second Tuesdays of January, March and May at 11 A. M. at the Weldon Hotel, Greenfield.

Middlesex East District Medical Society

January 14, 1931—Harvard Club of Boston.

March 11, 1931—At Reading.

May 13, 1931—At Wakefield.

Norfolk South District Medical Society

The meetings for the ensuing year are as follows: December 4, 1930, January 8, 1931, February 5, 1931, March 5, 1931, April 2, 1931, May 7, 1931.

All meetings begin at 12 noon. The February meeting will be held at the Quincy City Hospital. All other meetings will be held at the Norfolk County Hospital, South Braintree.

The censors will meet on May 7 at 11:30 A. M. at the Norfolk County Hospital, South Braintree.

NATHAN R. PILLSBURY, Secretary.

Suffolk District Medical Society

December 17-April 29—Combined meetings with the Boston Medical Library. These meetings will be held as specified below on Wednesdays, at 8:15 P. M.

December 17, 1930—Clinical Program, Boston Dispensary.

January 28, 1931—General meeting in association with the Boston Medical Library at the Boston Medical Library. "The Practice of Medicine in Relation to Public Health Activities." Speakers to be announced later.

February 25, 1931—Clinical Program, Boston City Hospital.

March 25, 1931—Clinical Program, Beth Israel Hospital.

April 29, 1931—Annual Meeting. Election of Officers. Boston Medical Library.

The medical profession is cordially invited to attend all of these meetings.

GEORGE S. DERBY, M.D.,
President, Suffolk District Medical Society.
LELAND S. McKITTRICK, M.D.,
Secretary, Suffolk District Medical Society.
HILBERT F. DAY, M.D.,
Boston Medical Library.

BOOK REVIEWS

The Doctor's Saddle-Bag. By JOSEPH GARLAND, M.D. The Massachusetts Linotyping Corporation, Boston, 1930. $1.50.

We liked the title and we liked the contents of these essays when they appeared from time to time in the *New England Journal of Medicine*, and we like them still more after reading them now collected in book form. This modern doctor-author with a sharp eye and a deep insight detects and diagnoses some of the social ills relating to medicine. He turns to his full saddle-bag, and instead of bringing forth an herb, a simple, a tourniquet or a bistoury, produces a logical and plausible cure for these ills. He treats our foibles with gentle sophistication, he tones up our interest and enjoyment of the everyday pleasures that come everyone's way, and then prescribes pleasant hobbies for the sluggish or bored, and new enjoyments out of simple things.

His diagnosis is searching and accurate, his treatments gentle or drastic as the case may demand, his advice understanding and reasonable, and his prescriptions, instead of being in antiquated Latin are written in modern English, and an English that is both distinctive and engaging.

Any physician, wearied by the technical descriptions of diseases, operations and therapies in his professional journals, may turn to these essays and find refreshment. And, if he stops to ponder as he reads, he may learn how to extract more of the true essence from life.

Others will find readable and enlightened discussions of the problem of the high cost of medical care, of health hazards, of the ways of doctors old and new, of various prohibitions, of the advantages of moderate means, and of the many varied aspects of this complicated and dizzy present-day life of ours.

These essays offer a solution for many a vexatious problem including a very present one. Especially to our readers and their friends with unfilled, perplexing Christmas lists before them, the reviewer, confident of thanks to be returned, recommends this "Doctor's Saddle-Bag."

A Syllabus of Lectures on Obstetrics for Nurses, prepared and published by a committee appointed by the American Gynecological Society. Copies may be obtained from the Chairman of the Committee, 23 E. 93rd St., N. Y. C.

In this little pamphlet of eleven lectures the committee of the American Gynecological Society is to be congratulated upon its effort to simplify the teaching of obstetrics to nurses. One wonders, however, what good this effort will do if certain of the important states continue to demand such absurd teaching for nurses as they now do.

The lectures are clearly and simply written, and for the greater part entirely according to the usual teaching. One or two careless statements have crept in, such as "the ileum, ischium and pubis, all being connected by strong ligaments", and the statement that "pelvic measurements afford some indication of the probable character of the labor".

It is, however, an excellent and praiseworthy attempt to simplify the teaching of obstetrics to nurses.